# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—33RD YEAR. SYDNEY, SATURDAY, OCTOBER 5, 1946. No. 14.

## Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.
Juvenile Rheumatism: A Survey from the Children's Hospital, Melbourne, with an Addendum on the Follow-Up System of the Almoners of the Hospital, by G. E. M. Scott, M.B., L.R.C.P. .. .. 469
Fibrositis and the Common Pains of Daily Practice, by Michael Kelly .. .. .. .. .. .. .. 480
Lumbago, by James H. Young, M.D., M.R.A.C.P. .. 485

REPORTS OF CASES—
Spinal Anæsthesia and the Pregnant Woman, by Arthur E. Brown .. .. .. .. .. .. .. 488
A Case of Exomphalos Successfully Treated by Early Operation, by George R. Hemming .. .. 490
Fibromyoma of the Small Intestine Causing Intussusception, by J. M. O'Connor .. .. .. .. 491

REVIEWS—
A Pocket Surgery .. .. .. .. .. .. .. .. 492
Clinical Neurology .. .. .. .. .. .. .. .. 492
The Causation of Appendicitis .. .. .. .. .. 492

LEADING ARTICLES—
The National Health and Medical Research Council 493

CURRENT COMMENT—
Beriberi Heart Disease .. .. .. .. .. .. .. 494
Vincent's Infection of the Gingivæ .. .. .. .. 495

ABSTRACTS FROM MEDICAL LITERATURE—
Gynæcology .. .. .. .. .. .. .. .. .. .. 496
Obstetrics .. .. .. .. .. .. .. .. .. .. 497

BIBLIOGRAPHY OF SCIENTIFIC AND INDUSTRIAL REPORTS—
The Results of War-Time Research .. .. .. .. 498

BRITISH MEDICAL ASSOCIATION NEWS— Page.
Notice .. .. .. .. .. .. .. .. .. .. .. 500

MEDICAL SOCIETIES—
The Medical Sciences Club of South Australia .. 500

NAVAL, MILITARY AND AIR FORCE—
Appointments .. .. .. .. .. .. .. .. .. 500

MEDICAL PRACTICE—
National Security (Medical Equipment) Regulations 502

POST-GRADUATE WORK—
The Melbourne Permanent Post-Graduate Committee .. .. .. .. .. .. .. .. .. 502
The Post-Graduate Committee in Medicine in the University of Sydney .. .. .. .. .. .. 503

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—
Queensland .. .. .. .. .. .. .. .. .. .. 503

NOMINATIONS AND ELECTIONS .. .. .. .. .. 503

OBITUARY—
John Richard Harris .. .. .. .. .. .. .. .. 504
John Charles White Halliday .. .. .. .. .. 504
Frank McCallum .. .. .. .. .. .. .. .. 504
Henry Pritchard Elliot .. .. .. .. .. .. .. 504

MEDICAL APPOINTMENTS .. .. .. .. .. .. .. 504

BOOKS RECEIVED .. .. .. .. .. .. .. .. .. 504

DIARY FOR THE MONTH .. .. .. .. .. .. .. 504

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 504

EDITORIAL NOTICES .. .. .. .. .. .. .. .. 504

## JUVENILE RHEUMATISM: A SURVEY FROM THE CHILDREN'S HOSPITAL, MELBOURNE, WITH AN ADDENDUM ON THE FOLLOW-UP SYSTEM OF THE ALMONERS OF THE HOSPITAL.[1]

By G. E. M. SCOTT, M.B., L.R.C.P.,
*Medical Clinical Assistant, Royal Melbourne Hospital, Melbourne.*

IN 1943 I published a review of rheumatic infection at the Children's Hospital, Melbourne, during the years from 1936 to 1942 inclusive.(1) This paper is a further contribution to the subject of rheumatic infection in childhood as seen at this hospital, together with some remarks on modern theories of causation, and the problem of prevention and control.

Experience has shown that the chief difficulties encountered are the diagnosis of the "insidious" case, and those patients complaining of "growing pains". Owing to shortage of staff during the war years, full use of investigational methods has not always been possible. Furthermore, convalescent treatment has been carried on under difficulty, owing to changes of location of the accommodation available.

The problem is well illustrated in the following two cases.

CASE I.—The patient, a boy, aged four and a half years, was admitted to the Children's Hospital, Melbourne, on May 24, 1946. On the previous day he had been examined in the casualty department of the hospital. It was noted that he had been well until three days previously, when he became "quiet and subdued", and was "off" his food. Two days before he was examined he had complained of pain in the left groin; he was unable to sit up, and screamed when moved. He also complained of pain in both legs, and would not move either limb. His temperature was found to be 101·6° F. Tenderness was located in both hip joints, in both knee joints and in the right ankle joint. On the day of his admission to the hospital his condition had improved somewhat, but pain was still present in the left hip joint, in the left knee joint and in the right ankle joint. Movement of the legs was much improved, but some abdominal pain was present. The temperature was 98·8° F., and physical examination gave negative results, but for a systolic murmur at the apex of the heart.

On May 30 the apex rate was 68 per minute, and the heart sounds were clear. Sinus arrhythmia was noted. There was no pain or swelling of joints, and no subcutaneous nodules were to be found. On May 31 a rather faint systolic murmur was heard at the mitral area, and an unduly accentuated second sound was evident at the pulmonary area. On June 3 this murmur was not audible, and the child's general condition had considerably improved. The erythrocyte sedimentation rate was 17 millimetres in one hour (Cutler method). The hæmoglobin value was 74%, and the leucocytes numbered 12,200 per cubic millimetre. The sleeping pulse rate was 80 per minute, and the temperature had fallen to 99·0° F. Sodium salicylate was administered as a routine measure.

CASE II.—The patient, a boy, aged six years, was referred to the Children's Hospital, Melbourne, by his local medical attendant on account of offensive stools, poor appetite and refusal to eat "fatty" foods. Inquiry revealed that he had been gaining in weight until five weeks previously, when his weight was 49 pounds. He had a normal appetite, and played well. However, his weight had dropped to 40 pounds, and he had lost energy. For the previous two weeks he had complained of pains in his legs, located around the knee joints, which had interfered with walking.

He was admitted to hospital on June 5, 1946, when it was found that his temperature was 98·4° F. On examination the apex beat of the heart was not well defined, but appeared to be just outside the nipple line in the fourth left intercostal space. The first apical sound was impaired, being soft; the second apical sound was unduly accentuated, as also was the pulmonary second sound. Examination

[1] Read at a meeting of the Melbourne Pædiatric Society on July 10, 1946.

gave otherwise negative results. The erythrocyte sedimentation rate was nine millimetres in one hour, and the hæmoglobin value was 66%. The leucocytes numbered 8,850 per cubic millimetre. In seven days from the time of his admission to hospital the patient's condition was much improved. The mitral systolic murmur was more definite, and the apical second sound was not unduly accentuated. Sodium salicylate was administered as a routine measure.

It will be noted that both Case I and Case II were mild. In neither was there a family history of rheumatic fever, nor any note of sore throat preceding the onset of the rheumatic episode. Neither patient exhibited polyarthritis, and an Case II the "growing pains" were accompanied by loss of weight and of energy together with anæmia. By contrast, the case of an adult patient is presented. In this case it will be observed that sore throat was accompanied by transient arthritis, and the diagnosis suggested was one of early rheumatoid arthritis, or possibly subacute nephritis.

CASE III.—The patient, a female, aged twenty-three years, was admitted to the out-patient department at the Royal Melbourne Hospital on May 18, 1946, under the care of Dr. J. H. Bolton. She had been referred by her local medical attendant on account of intermitent swelling of the ankle joints for the previous two months. Salicylates had been administered without much improvement. No obvious focus of infection was found. The tonsils had been enucleated some time previously. There was no history of trauma. Otherwise she was quite well. The past history was clear. On examination, no abnormalities were found. Radiological examination of the ankle joints gave normal results.

On May 31, 1946, the patient presented herself, complaining of sore throat. Both ankle joints had been swollen and painful for the preceding twelve hours. The right ankle joint was a little œdematous, and some pain was present on movement. The throat was a little injected. Otherwise examination gave negative results. A catheter specimen of urine yielded no growth on attempted culture. An occasional epithelial and pus cell and granular cast were found. The erythrocyte sedimentation rate was 5·5 millimetres in one hour. Attempted culture from a throat swab produced no hæmolytic streptococci. As on previous occasions, the swelling of the ankle joints subsided in the course of a few days.

### Ætiology.

The general consensus of opinion tends to associate acute rheumatism with the hæmolytic streptococcus. Coburn (1931)[3] and others have attempted to show the relationship on clinical and bacteriological grounds. The question of allergy and immunity has been pressed more vigorously in recent years. The epidemiological factor is also of considerable importance.

The problem of bacterial allergy is well summarized by Hadfield and Garrod,[3] and the example of tuberculosis well illustrates the essential conceptions. Animal experiments show that sensitization to the tubercle bacillus results in a local inflammatory reaction on injection of tuberculin, and local necrosis may result. The animal has acquired the capacity to respond by a local acute inflammatory reaction. This reaction differs from that of anaphylaxis, which results after injection of a foreign protein. In anaphylaxis spasm of smooth muscle occurs, and furthermore, anaphylaxis can be transferred passively from one animal to another, whereas the serum of one animal cannot transfer the allergic reaction to another animal. In anaphylaxis antibody is present in the circulation; but in the allergic state no antibody is formed. According to Rich and Lewis[4] and to Aronson,[5] the allergic state consists in changes in the cells themselves. The followers of this theory believe that hypersensitivity or the allergic reaction is an incidental and chiefly harmful reaction, bearing no relation to immunity in the host. Others hold that the allergic state implies a protective mechanism for dealing effectively with the infective process. Opie[6] contrasted the pulmonary lesions of tuberculosis in children and in adults, and showed that in the former the lesions tended to be more widespread, whereas in the latter the pathological processes were more localized. The experimental work of Krause[7] and Willis[8] tends to support this view, and to attribute localization of the infected area to the operation of allergy. Rich,[9] and Rich and McCordock,[10] however, maintain that wide dissemination of tubercle bacilli in young subjects is due to lack of immunity, and that the adult must suffer a bacteriæmia which is not evident on account of destruction of the bacilli by resistant tissues throughout the body. While these theories have been argued on the question of infection by the tubercle bacillus, the same may be applied to any infection. It is generally accepted that persons yielding a positive skin reaction to tuberculin are less likely to develop active tuberculosis than those who fail to react, and that the allergic response operates by localizing such infection.

In 1932, Collis, Sheldon and Hill[11] published a paper on the cutaneous reactions in acute rheumatism, and summarized the literature on the subject and on the question of hæmolytic streptococci in relation to rheumatic infection. They called their test substance "H.S.E." (hæmolytic streptococcal endotoxin); this was an endotoxin prepared from hæmolytic streptococci. They concluded that "rheumatic" children were more sensitive to this substance than were non-rheumatic children, and that children with chorea were more sensitive than those with carditis. Approximately 80% of rheumatic children gave strongly positive reactions for a period up to six months after their last acute attack, whether this was accompanied by chorea, carditis or polyarthritis. After a period of six months the reaction diminished. Age was found to be an important factor in the matter of reaction up to puberty, and children acutely affected failed to react. Collis, Sheldon and Hill state that the "H.S.E." may be compared to tuberculin in its mode of action, and also in other properties such as heat stability. In contrast to the response to "H.S.E.", however, the response to the tuberculin test tends to remain positive. Furthermore, the failure to occur of the "H.S.E." reaction in acutely ill rheumatic children, and its return on subsidence of the infective process, run parallel with the same phenomenon in children suffering from acute tuberculosis, in whom the Mantoux test may fail.

Gibson, Thomson and Stewart[12] studied intradermal reactions to extract preparations of streptococci of varying serological types. They concluded that strongly positive reactions to hæmolytic streptococcus extract were more common in rheumatic subjects than in control subjects. Skin sensitivity was found to increase up to the age of fifteen years, and thereafter there was no further increase in sensitivity. Moreover, the skin reactions might be the result of previous streptococcal infection, but were not regarded as of value in diagnosis or prognosis in rheumatic infection.

Taran, Jablon and Weyr[13] also studied the cutaneous reactions, using the "M" fraction of hæmolytic streptococcus. Their patients included the normal siblings of rheumatic children, and normal children, as well as rheumatic children. A positive cutaneous reaction was obtained in 65% of normal children and in 83% of rheumatic children. The normal siblings of rheumatic children gave the same reactions as did rheumatic children. In normal children the reaction was significantly milder in degree. These workers state that the varying percentages may not be statistically significant. They regard the "M" fraction as primarily associated with type-specific antibody formation. The "M" fraction is regarded relatively as purely protein in nature.

Coburn and Pauli[14] believe that subjects of rheumatic fever who suffer transient respiratory infections produce antibodies to hæmolytic streptococci for some months after subsidence of the attack. They collected the serum of rheumatic subjects at weekly intervals after the onset of pharyngitis for a period of three to six months. Sixteen patients were reported on in one of their studies. Each sample of serum was tested for antistreptolysin, for Lancefield's type-specific precipitins, and for specific agglutinins by the Griffith slide agglutinin technique for the typing of hæmolytic streptococci. Cultures of the organism recovered from the patient's throat during the acute phase were extracted, and type-specific "M" substances were prepared from these for the precipitin tests. In the slide agglutinin tests, suspensions of the patient's organism, and control suspensions of other serological types, were used against the serum. Coburn and Pauli's finding in this study pointed to a prolonged activity of the hæmolytic streptococcus as a whole. In all of the 16 cases there was a striking rise in

antistreptolysin titre and also in type-specific antibodies. In fourteen, precipitins were found and in eleven, slide agglutination reactions. Coburn and Pauli state that in the majority of cases antibody to the "M" substance continued to increase after the antistreptolysin titre had reached its maximum level. Thus both antibodies were affected in any prolongation of the immune response, and not antistreptolysin alone. Coburn and Pauli consider that their findings support the view that prolonged increases in streptococcal antibody, such as are observed in rheumatic fever, signify subclinical activity of hæmolytic streptococci.

Rantz, Kirby and Jacobs[15] come to the following conclusion: "While the presence of type specific agglutinins for the hæmolytic streptococcus may be an indication of the presence in the individual, of antibacterial immunity, the presence of these antibodies is not sufficient to guarantee resistance to infection."

Wilson and Lubschez,[16] using statistical methods, claim to have shown that the recurrence rate of rheumatic infection is greatest during the first year after a major episode. A major episode is defined as arthritis, chorea and active carditis, alone or in combination.

An analogy may be drawn between the initial attack of rheumatic infection and the primary infection of tuberculosis. In this way the recurrence in rheumatic fever would be following a changed pattern, due to the operation of allergic and immunological factors, at present incompletely understood. It is possible, too, that many cases of rheumatic infection, on account of their mild nature, are overlooked, and thus patients are examined in what may actually be a recurrence of infection. This recurrent infection may at times have maximal incidence in and be localized to the heart, and this may account for permanent damage to that organ.

Bland and Jones[17] conducted a ten-year follow up study of 314 children and adolescents with potential rheumatic heart disease and reached the following conclusions:

1. Characteristic signs of permanent valvular deformity, not present at the time of the original illness, appeared later in 79 patients (25%).

2. The so-called delayed appearance of heart disease was clearly associated with coexisting signs of recurrent rheumatic fever in two-thirds of the group.

3. In the remaining one-third heart disease appeared insidiously. Certain evidence suggests that here also the appearance of the signs of heart disease coincided at this age with persistent subclinical rheumatic activity.

4. Further observation into the second decade indicated that a sharply diminishing incidence in the later appearance of heart disease was to be expected in the remainder of the patients.

### Pathology.

The essential lesion of acute rheumatism was described by Aschoff[18] in 1906. Von Glahn and Pappenheimer[19] also published a comprehensive study in 1926. There is an extensive literature on the subject, and it is possible here only to refer to recent work.

John D. Robinson,[20] from an experimental study, reached the following conclusion: "Rheumatic fever is considered a disease consisting essentially of an autogenous antigen-antibody reaction involving connective tissue collagen and streptococcus toxin." Robinson's experiment was made with young albino rabbits, and although typical uncomplicated rheumatic fever was not produced, many lesions typical of human streptococcal disease and some cardiac lesions closely resembling the human type resulted. A similarity is drawn to anaphylactic or allergic phenomena. Body tissue combines with toxin to form antigen. There follows a period of formation of antibodies against the tissue *plus* toxin complex, and when the titre is sufficiently high an autogenous antigen-antibody reaction occurs. Delayed serum sickness is a good example of such a reaction. If rheumatic fever is viewed in this light, its undoubtedly infectious nature is explained and a link with streptococcus infection is suggested. This explanation also covers its non-contagious nature and the singular pathology of collagenic alteration. The question arises why the high antibody titres in rheumatic subjects do not prevent absorption of toxins and combination with body tissues. This is probably because toxins combining with body tissue are of non-antigenic (hapten) nature. The further question then presents itself, why are antiodies formed against the patient's own tissues? Robinson believes this is because the tissues are now altered through combination with hapten toxins. Robinson states that "fundamentally there is probably an inherited tendency to a tissue weakness of this type". Robinson's hypothesis recalls Coburn's theory of the body dealing with streptococcal products in a "peculiar way".

Rich and Gregory[21] and others[22,23,24,25] have also conducted experiments in order to elucidate the essential pathology of rheumatic infection. Green[26] and Collis,[27] and Scott Thompson and Innes[28] examined fresh autopsy material.

Wright-Smith[29] reached the following conclusions. There is a toxic spoiling or cloudy swelling of the cells of the myocardium. Cell infiltration occurs between the muscle fibres. Changes are present in the smaller arterial vessels, and these are characteristic of a non-specific lesion. In larger vessels typical rheumatic lesions are found. The coronary arteries may be involved at their orifices, and thus anoxæmia of the cardiac muscle may result. Wright-Smith believed that the lesions found in the aorta resemble those due to syphilitic infection and occur at the same site. However, no aneurysm formation occurs, because the media of the aorta is not sufficiently affected in rheumatic infection.

No account of the pathology of rheumatic fever would be complete without some reference, however short, to the vexed question of rheumatic "pneumonia", or pneumonitis, as some prefer to call it. As long ago as 1888, Cheadle[30] described an outbreak of this affection in the previous year. Cheadle noted that "pneumonia sometimes does occur in the course of acute rheumatism", and he mentioned that there was a wide difference of opinion as to the frequency and importance of the affection. He also noted the appearance of the pneumonic process in the lower lobe of the left lung, the absence of typical cough, rusty sputum, and the fall of the temperature by lysis and not by crisis. Cheadle observed the close association of pericarditis, and the time interval of some days between the occurrence of pneumonia and that of pericarditis. In four out of six cases described by Cheadle, old standing valvular disease was present. To account for the difference in symptoms and signs found in the lung affection associated with acute rheumatism, Cheadle considered that the air cells and bronchioles must be in a different state. On the authority of one, Sturges, who wrote a monograph on pneumonia at the time, Cheadle suggested that the pulmonary consolidation might be due to "a rapid transudation of fibrin from blood in a highly fibrogenous condition circulating under conditions of special mechanical difficulty". Thus a defect of the mitral valve would cause obstruction to the venous return from the lung, and pericarditis would cause serious embarrassment to the action of the heart, as myocarditis would be responsible for weakening of the muscular action of the heart. Cheadle recognized, however, that all such observations failed to explain the transfer of incidence from the right lung to the left lung. It has been suggested that pressure of an enlarged pericardial sac, together with inhibition of the left hemidiaphragm, may result in a partial collapse of the lower lobe of the left lung. If this explanation is accepted as a partial explanation of the phenomena, then it becomes unnecessary to seek for the presence of Aschoff bodies in the lung tissue in order to prove that the process is a rheumatic manifestation. For when the Aschoff bodies are not to be found, the further postulate must be made that, owing to the physical texture of the lung tissue, these structures are so altered as to be unrecognizable as such, and this appears to be a contradiction in terms. The one essential fact of importance is that the "rheumatic" lung is constantly associated with pericarditis. The ancillary observation is that endocarditis and probably myocarditis are universally found in patients suffering from "rheumatic" pneumonia. It follows that a mechanical factor cannot entirely be ruled out, and it need only be added that a patient with pericarditis due to acute rheumatism may suffer an attack of pneumonia on the right lung, and that this disease on the right side is modified by the presence of rheumatic pericarditis. Such an explana-

tion, while it may not account for all the observed facts, leads to less confusion of thought than one which endeavours to confuse the issues.

I have seen one instance of lobar pneumonia, involving the lower lobe of the right lung in a child, aged eight years, suffering from rheumatic pericarditis. The patient developed a sudden severe pain in the right lower part of the thorax posteriorly, with some dyspnœa and a rise of temperature to 104° F. On examination of the child some six or seven hours later, some impairment of the percussion note was found at the area of the base of the right lung, together with diminution of the vesicular murmur and prolongation of and rise of pitch in expiration; but no adventitious sounds were audible. Sulphamerazine had been administered in full therapeutic doses at the onset of the pain in the chest, and in the course of twenty-four hours the temperature had subsided to 99° F., and the patient seemed more comfortable. The subsequent course of the pneumonic process was uneventful. No moist sounds appeared in the affected area, and the breath sounds returned to normal in the course of the succeeding two or three days. No radiological examination was made. The pericarditis from which the patient was suffering was subsiding at the time of onset of the pulmonary complication, and no abnormal physical signs were to be found at the base of the left lung.

The problem of the "rheumatic" lung has been studied by Naish,[31] by Fraser,[32] by Finlay,[33] and by Coburn[34] and others.[35][36][37][38][39]

## Early Diagnosis of Rheumatic Infection.

### *Onset.*

Three phases are recognized in the development of an attack of rheumatic fever. The first is an acute upper respiratory tract infection, lasting for a few days. This phase may be transient and may go unnoticed on account of its mild nature. In the second phase, regarded by Coburn as the crucial phase, there is complete absence of clinical symptoms and signs. This period usually lasts for one to three weeks, but may last for a few days to six weeks. In the third phase the rheumatic manifestations, varying widely in intensity and duration, make their appearance. It is recognized that streptococci are closely related to acute rheumatism, and it should be remembered that these organisms are essentially parasites of the throat, and that the $\beta$ hæmolytic streptococcus may be found in the throat apart from the incidence of acute rheumatism. The subject of streptococcal throat infections and the relation to tonsillar tissue, has been studied by Keogh, MacDonald, Battle, Simmons and Williams,[40] and by Elliot,[41] and by MacDonald, Simmons and Keogh.[42]

### *Polyarthritis.*

Polyarthritis is widely described and well known, and does not require detailed description in this paper. Some difficulty is occasionally encountered in distinguishing arthritis and osteomyelitis. The distinction is important in early diagnosis of acute rheumatism, as sulphonamides will be ineffective in this condition, whereas acute osteomyelitis may be relieved by their administration. During an epidemic of poliomyelitis, limb pains and the attendant immobility may lead to acute rheumatism being overlooked. The reverse problem obtains in the absence of an epidemic of poliomyelitis. Acute epiphysitis may occasionally cause difficulty. Rheumatic fever has occasionally been encountered in children under the age of three years. In my own series of 645 cases, polyarthritis was found to be present in 20·15%.

### *"Growing Pains."*

The old idea that growth is painful arose from a theory that vascular congestion of the epiphyses caused discomfort. Jacobi, who is mentioned by Castiglioni[43] in his book "A History of Medicine" as "the virtual founder of pediatrics in America", has been credited with the doubtful honour of having originated the term "growing pains". If the term "growing pains" could be abandoned and forgotten by clinicians, a great service would be rendered to the study of acute rheumatism. However, it would be optimistic to imagine that this could be attained with a phrase so deeply rooted in pædiatric literature. "Growing pains" occurred in 60·4% of my series of cases. It does not necessarily follow that acute rheumatism was the cause in every case. Shapiro[44] compares the symptoms and signs of "growing pains" and subacute rheumatism. Shapiro's main points are that "growing pains" occur in early childhood, and continue through to adolescence, whereas subacute rheumatism is most commonly found between the ages of six and seven years, and often follows upper respiratory tract infections and other infectious diseases. Pains of non-rheumatic origin are complained of at the end of the day, and especially during the night; in the morning the pains have disappeared. By contrast, the pain in subacute rheumatism occurs when the patient first gets out of bed in the morning, and may last all the succeeding day, being more troublesome on movement. A limp may be noticed, and relief is afforded by warmth and rest in bed. While the pain of subacute rheumatism is localized in the joints, non-rheumatic pains are felt in the muscles of the legs and in the thighs. Furthermore, laboratory findings, such as the erythrocyte sedimentation rate, the leucocyte count and hæmoglobin level, are normal in the patient complaining of "growing pains". Hawksley[45] also discusses the nature of "growing pains" and their relation to rheumatism in children and adolescents. Hawksley believes that non-rheumatic pains are located in muscles, ligaments and tendons, and that tenderness is rarely to be found on examination. He mentions postural and orthopædic defects as associates of "growing pains", and refers to the sulky and unsociable nature of children so afflicted. Subcutaneous nodes are absent in patients who complain of "growing pains". Anæmia is characteristic of subacute rheumatism. Hawksley finds the incidence of carditis subsequent to "growing pains" to be not greater than would be expected to occur in the child population generally. From personal experience, I can fully support the opinions of both Shapiro and Hawksley. I believe that no child should have the label of acute rheumatism attached on the ground of "growing pains", in the absence of some supporting evidence of a definite nature. I further consider that many children have been needlessly confined to bed, or had their normal activity checked by failure to recognize the observations which have been stated. Still thus aptly puts the position: "A common antecedent of heart disease is this complaint of 'growing pains', which so often represent acute articular rheumatism in children." Still[46] then goes on to say how very important it is to remember that these vague aches and pains "may be associated with just as severe endocarditis as the most acute attack of so-called rheumatic fever". Sometimes it is necessary to abandon the dicta of renowned physicians, or to read a new meaning into their writings, if progress in the study of medicine is not to be hampered. It seems to be clear, from a careful reading of Still's account of rheumatism, that he was in a difficulty in interpreting the significance of "growing pains" in cases in which the joint phenomena were absent. Writing of the statements of parents that their children had "only 'growing pains' ", he makes the following statement: "Allowing however for such a fallacy, one sees cases of undoubted rheumatism in childhood in which joint phenomena would seem to be entirely absent." Still probably sought some lead in diagnosis, and consequently the impression was given that "growing pains" were important in themselves, which is clearly not the true position. Jacobi[47] wrote: "The large majority of attacks of growing pains means rheumatism." But Jacobi also wrote that in many instances he considered growing pains to be "but the expression of muscular fatigue". Perhaps he, too, was puzzled by the discovery of serious heart disease in the absence of any definite history of illness in his child patients.

### *Cardiac Murmurs.*

Systolic murmurs are frequently heard at routine examinations of the heart in childhood. In the absence of suspected congenital lesions, these murmurs are frequently of no significance. A systolic murmur, heard close to the sternum in the mitral area, may generally be neglected. From a wide experience, Grieve[48] believes such murmurs are not associated with cardiac disease. Gibson[49] states that "heart murmurs are so frequent during the period of childhood that even in a well child the absence of a murmur rather than its presence occasions surprise". I believe that much confusion can be avoided

if mitral systolic murmurs which are faintly heard are described as "faint" and not as "soft" murmurs. The word "soft" ought to be reserved for a description of the impaired first sound at the apex area, which is heard in early affection of the mitral valve, and which is probably due to myocarditis. This murmur is a most important sign to elicit in early acute rheumatism. Gibson discusses the systolic murmurs heard at the second left intercostal space over the pulmonary area, and the systolic murmur heard medially to the apex at about the third left intercostal space. Gibson then states that when these murmurs are audible "only on careful auscultation one can feel reasonably confident that they are without significance". I believe that systolic murmurs heard faintly anywhere between the apex beat and the left sternal border in convalescent rheumatic children should be ignored in the absence of satisfactory evidence of activity of rheumatic infection. These murmurs are of no significance in children with a past history of acute rheumatism who feel well and enjoy normal activity. Ritchie[50] wrote as follows of acute rheumatic carditis: "It is true that in some cases those lesions must be transient, because life assurance companies subsequently accept those lives as first class and old age is attained in the enjoyment of good health." The following signs, described by Coombs,[51] are a most useful guide in the early diagnosis of acute rheumatism: (i) evidence of cardiac enlargement to the left; (ii) impairment of the first apical sound—that is, a "soft" bruit; (iii) accentuation of the second apical sound; (iv) an unduly accentuated pulmonary first sound. Of these early cardiac signs, the second is probably of most significance; for the first, enlargement of the heart, as evidenced by displacement outwards of the apex beat, may not be evident at the first examination.

### *The Insidious Case.*

In my 1943 series of 645 cases, chorea was found to be present in 25·8% and acute polyarthritis in 20·15%. The remaining 54% (54·05%) evidently presented some problems in diagnosis. The insidious case of acute rheumatism, because it is frequently missed in diagnosis, is probably responsible for much of the cardiac disease discovered only in later life. Every child who may be suspected of suffering from rheumatic fever, or in whose case such a "provisional" diagnosis is made, should be investigated with all the care and all the aids available in order to arrive at a correct diagnosis. Finality should be reached if possible within a period of fourteen days from the first examination of the patient. If the diagnosis of acute rheumatism is not made, then the child should at once be allowed normal activity. The following points may be found to be a useful guide in diagnosis:

1. A carefully recorded history of the case. First, the family history appears to be of some importance. In my series of patients, a family history of rheumatic fever was elicited in 24·49%. Of this figure, the closest contacts of the patient, the mother and the siblings, comprised 6·51% and 7·28% respectively. The remaining 10·70% comprised the uncles, aunts and grandparents of the patient. The second observation should be the occurrence of other illnesses, such as scarlet fever, nephritis and tonsillitis. Finally, note should be taken of any complaint of lassitude, loss of weight, pallor or aching limbs on rising in the morning, and in the case of young children refusal or inability to use the limbs freely.

2. In addition to cardiac signs mentioned, the presence or absence of sinus arrhythmia. A careful record of the heart rate at the apex and of any change in the character of the cardiac murmurs from day to day is important.

3. A thorough search for subcutaneous nodes.

4. The temperature charts, and records of alert and sleeping pulse rates. These are important additional aids.

5. The use of investigational methods. These are essential, and of them estimation of the erythrocyte sedimentation rate (if the temperature is normal); of the hæmoglobin value; and of the number of leucocytes is most useful. Electrocardiographic tracings may sometimes be of service in insidious cases, by revealing a prolongation of the auriculo-ventricular conduction time.

The finding of sinus arrhythmia on listening at the apex of the heart may be of some importance, but its absence has much less significance. Mackenzie,[52] in discussing the prognosis of disturbances of the sino-auricular node, referred to an irregularity of the pulse which he termed the "youthful" type of irregularity. Mackenzie wrote: "In cases where the heart might be affected, as in rheumatic fever, I observed that the appearance of this irregularity was an indication that the heart had escaped damage, even although a systolic murmur may have developed during the illness."

### *The Erythrocyte Sedimentation Rate.*

Determination of the blood sedimentation rate is essential to effective control of convalescence in acute rheumatism, and is frequently useful in early diagnosis of the disease. Fahreus, discussing the suspension stability of the blood, writes: "There is probably no observation in the history of medicine which has played such a big part as has this phenomenon." It has the closest association with blood letting, and was called the *crusta sanguinis*, "buffy coat" or "blood cake". It was the superficial layer appearing on the clotted blood left after blood letting, and represented a rapid sedimentation of the erythrocytes. This phenomenon was recognized over two thousand years ago as occurring in diseased conditions. The authors who have contributed to the study of the erythrocyte sedimentation rate in disease may be divided into two classes—namely, those who have directed attention to the investigational side, and those who have considered mainly the clinical applications of the test. In the former class are Fahreus,[53] Westergren,[54] Ernstein,[55] Zeckwer and Goodell,[56] Peterman and Seeger,[57] Landau,[58] Wintrobe,[59] Beaumont and Maycock,[60] and Hynes and Whitby.[61] On the clinical side are Payne,[62] Cutler,[63] Bach and Hill,[64] Struthers and Bacal,[65] Perry,[66] and Payne and Schlesinger.[67]

Ernstein made observations on the sedimentation rate in conjunction with the estimation of the plasma fibrinogen content and the leucocyte count. He found the plasma fibrinogen content to be increased in rheumatic fever and in other infections, and found also that there was a close relationship between the concentration of this protein and the erythrocyte sedimentation rate. He regarded these observations as of more value in rheumatic fever than the leucocyte count.

Zeckwer and Goodell made use of a centrifuge tube, and measured the height of the red cell column. They found an increased rate in a variety of conditions, including acute inflammatory conditions, malignant tumours and tuberculosis. Zeckwer and Goodell considered that an increased rate bore a relation to destruction of tissue.

Peterman and Seeger consider that the erythrocyte sedimentation rate has little value in diagnosis, and no value in prognosis. They state that "a single normal or slightly increased or decreased rate means little. A single rapid rate means abnormality". Frequent leucocyte and differential counts have been of much more assistance in following the clinical course of disease. Peterman and Seeger add a voluminous list of references.

Landau refers to the difficulty of venepuncture in corpulent people, small children and babies, and to fear of the operation in young children. Landau regards normal values as not exceeding five or six millimetres in one hour, but does not draw too narrow limits. He states that in patients up to two years of age the rate is one to five millimetres in one hour, and in those over two years of age it varies from one to eight millimetres in one hour.

Wintrobe uses the hæmatocrit method. Beaumont and Maycock compared the Westergren method, the micro-method, and that of Zeckwer and Goodell, and concluded that the value of the test lay in the comparison of readings by the same method, from week to week. Hynes and Whitby give a chart recommended for correction of the sedimentation rate for anæmia. As the anæmia of rheumatic infection is maximal in the iron content of the erythrocytes, and not in the number of red cells, this correction is not required.

Payne describes a method in which only 0·4 millilitre of blood is used, and correlates this with clinical findings.

Cutler conducted observations on approximately five thousand patients over a period of six years. He gives

a useful table showing the relative effects of various diseases on the sedimentation rate.

Bach and Hill state that in juvenile rheumatism in the active phase the blood sedimentation rate is raised. In quiescent rheumatism or rheumatic heart disease without clinical signs of activity the rate is near normal limits. The test may be of real value in the diagnosis of doubtful cases. Bach and Hill took the normal to be below 10 millimetres at the end of the first hour, and below 20 millimetres at the end of the second hour, using the Westergren method. They consider that if the erythrocyte sedimentation rate is persistently low, there will be little danger in allowing the child the resume normal life.

Struthers and Bacal conducted an investigation, using Payne's technique, and observed a control group. They found that in uncomplicated acute rheumatic fever the sedimentation rate was high, that usually a leucocytosis of 12,000 to 15,000 per cubic millimetre was present, and that approximation of the sleeping and waking pulse rates during the period of fever and loss of weight occurred. They also found that "chorea without carditis showed no alteration in the total white blood cell count, sedimentation rate of fever"; but that the sleeping pulse rate was normal, and there was usually a marked difference between it and the alert pulse rate. Except in the presence of cardiac failure with œdema, the sedimentation rate is probably the most delicate test of activity of the infection.

Perry considers that acute rheumatic carditis is always associated with a high blood sedimentation rate. Although active carditis is unlikely in the presence of a high sedimentation rate, repeated observations should be made in polycyclic cases. Should congestive cardiac failure supervene, the rate returns to normal. In chorea unaccompanied by carditis the rate may be normal.

Payne and Schlesinger studied the blood sedimentation rate in relation to various infections such as acute tonsillitis, chronic tonsillar sepsis, tuberculosis and rheumatoid arthritis. They state that "the close relationship between rheumatic activity and the sedimentation rate has been confirmed". With regard to a diagnosis of acute rheumatism, Payne and Schlesinger consider that the temporary rise of the rate found in acute tonsillitis and influenza must be taken into account. In acute chorea there may be a small temporary rise of the rate, or a rise may even be absent, and here no evidence is given of possible subsequent carditis. The rate may fall to normal in congestive cardiac failure, even in the presence of active disease, and this is a bad prognostic sign.

### *Sleeping Pulse Rate.*

The record of the sleeping pulse rate has been found most useful in all stages of the management of rheumatic infection. It should be assessed in conjunction with other signs, and must be carefully recorded by a nurse who has been specially instructed. The normal alert and sleeping pulse rates vary according to age of the child. In older children the sleeping pulse rate is 10 beats per minute lower than the alert pulse rate. Schlesinger[69] conducted a comprehensive study of sleeping and alert pulse rates in rheumatic children, and Table I illustrates the result of his investigation.

As an illustration of the value of records of the alert and sleeping pulse rates in the case of excitable children, the following case from Schlesinger's paper is quoted.

A girl, aged six and a half years, had had complete bed rest for six months on account of pyrexia to 99° F. and a pulse rate varying from 90 to 110 beats per minute. The sleeping pulse rate was recorded and found to be normal and to be 22 beats per minute below the alert rate. No evidence of cardiac disease was found on examination, and the child was allowed to get up. She is reported to have remained well.

### *Hæmoglobin Value.*

The hæmoglobin value is regarded as a most useful observation, both in the early case and throughout convalescent treatment. Osgood and Baker[69] give the following normal values: boys aged from four to thirteen years, 80·7% (11·14 grammes *per centum*) to 93·4% (12·89 grammes *per centum*); girls aged from four to thirteen years of age, 88·5% (12·21 grammes *per centum*) to 93·6% (12·92 grammes *per centum*). The figures of Mackay, Wills and Bingham[70] agree substantially with these.

### *Leucocyte Count.*

Counting of the leucocytes is not regarded as a very reliable observation in the diagnosis and treatment of acute rheumatism, owing to normal fluctuations incidental to childhood. Sheldon[71] makes the following statement: "Within a day or two of birth the white cells number up to 25,000 per cubic millimetre. By the end of the first year they have fallen to 15,000 per cubic millimetre of which 60 to 65 *per cent.* are lymphocytes. By the fifth or sixth year the count has fallen to about 10,000 per cubic millimetre of which about 40 *per cent.* are lymphocytes." Coombs was not impressed by the value of this test.

TABLE I.
*Normal Alert and Sleeping Pulse Rates.*
(*After* Schlesinger, Modified.)

| Age. | Alert Rate. (Per Minute.) | Sleeping Rate. (Per Minute.) | Difference. |
|---|---|---|---|
| 0 to 12 months .. | 132 to 120 | 128 to 113 | 4 per minute. |
| 1 to 10 years .. | 107 to 85 | 99 to 77 | 7 or 8 per minute. |
| 10 to 15 years .. | 85 | 75 | 10 per minute. |

### *Electrocardiography.*

The electrocardiographic tracing is not regarded as generally applicable in acute rheumatic fever. It may, however, be of service in some cases in which the diagnosis is in doubt. The observation of importance is the auriculo-ventricular conduction time. Elghammer[72] has recently conducted a survey in which use was made of this test. Pardee[73] and Evans[74] have written on the subject. Pardee states that the *P-R* interval of the electrocardiogram varies with the heart rate, being shorter at more rapid rates, and longer at slower rates. Pardee observed that the *P-R* interval tended to be shorter in childhood, varying between 0·12 and 0·20 second.

### *Body Weight.*

The body weight is considered a reliable index of progress during convalescent treatment and in the follow-up system at the Children's Hospital, Melbourne.

## Clinical Aspects.

The following case illustrates an attack of rheumatic fever of moderate severity preceded by a history of "growing pains", which presented no particular problem in diagnosis. There was one recurrence, and the heart apparently escaped permanent damage.

CASE IV.—E.W., a female patient, aged six years and seven months, was admitted to the Children's Hospital, Melbourne, on April 12, 1943. Investigation of the family history revealed that her mother had complained of "rheumatics" in her toes and ankles six years earlier. Her father was alive and well, and there were two other children, both well. The previous history revealed that the patient had had chorea and occasionally became hysterical in her sleep. She had had muscle pains six years earlier and five months earlier, and she had suffered from an attack of tonsillitis six years previously without apparently any ill effects. She had complained of being off colour and tired for the previous three days. Her right knee joint had been painful and swollen and she had difficulty in walking. On the morning of her admission to hospital she complained of swelling and tenderness in the right ankle joint. The condition of the right knee joint had improved. On examination of the patient the apex beat of the heart was in the fourth left intercostal space two and a half inches from the mid-sternal line in the nipple line. The heart sounds were clear and the presence of sinus arrhythmia was noted. The right ankle joint was slightly swollen and the right knee joint was affected by slight periarticular swelling, but there was no pain on movement. Physical examination otherwise gave negative results. Complete bed rest was ordered without the use of a pillow; but the patient was allowed to feed herself. Sodium salicylate in doses of 15 grains with 30 grains of sodium bicarbonate was administered every four hours.

On April 24 the joints were found to be normal, but the patient complained of sore throat. On examination the

throat was seen to be injected, and culture from a swab revealed the presence of hæmolytic streptococci. The heart was normal, and she was given sulphanilamide, 7·5 grammes (one tablet) every four hours, and the administration of sodium salicylate was continued in doses of 10 grains with 20 grains of sodium bicarbonate three times a day. With the onset of the throat infection, the sleeping pulse rate had risen from 76 to 88 beats per minute. By May 4 the throat condition had subsided and sulphanilamide treatment was discontinued. The administration of sodium salicylate was continued in the previous dosage. The results of examination were otherwise normal, and the child was transferred from the hospital to the convalescent hospital.

On May 17 the administration of sodium salicylate was discontinued. The patient was still ordered complete bed rest.

On May 24 the patient was allowed up for half an hour in the morning. A week later, on May 31, the sleeping pulse rate had been steady at 80 beats per minute, and physical examination gave normal results. A further half-hour per day out of bed was allowed, so that she was up for one hour per day. The child was difficult to control in bed and rather non-cooperative, and as her condition was good she was allowed up for two hours each day by June 7.

On June 14 examination revealed no abnormality, and she was allowed up for four hours per day. She was then discharged home, and instructions were given that she should be allowed up for two hours each day, this amount to be increased by half an hour each week until she was up for three hours per day, when she was to attend the rheumatic clinic at the hospital. She attended the rheumatic clinic on July 3, having been well since her discharge from the convalescent hospital. Physical examination revealed an unduly accentuated pulmonary second sound, but no other abnormality. Instructions were given that she was to be allowed out of bed for one and a half hours in the morning and for one and a half hours in the afternoon. By August 14 she was up all day.

On August 18 she was readmitted to the hospital complaining of pain and swelling in both knee joints and ankle joints, present for the previous four days. On examination of the child, her temperature was 99·4° F. and the pulse rate was 120 beats per minute. The apex beat of the heart was in the nipple line, and a soft systolic murmur was audible at the tricuspid area. The pulmonary second sound was unduly accentuated. There was gross swelling of both knee joints and ankle joints, which required the application of methyl salicylate foments. Sodium salicylate was administered as a routine measure. Routine treatment was continued until October 4. The systolic murmur was still audible at the tricuspid area, but examination revealed no other abnormality. She was up for two hours twice a day. She was then discharged from the convalescent hospital, to which he had been admitted on August 24.

She reported at the rheumatic out-patient clinic on December 4. Examination at this visit revealed no abnormality. She was instructed to be up for four hours each day, and to increase the time up by half an hour each week.

On January 8, 1944, she was again examined. She had been well in the intervening period, but had a temperature of 101° F. Examination otherwise revealed no abnormality, and she was referred to the casualty department of the hospital for observation and admission if necessary. She attended again on January 15, when the temperature was found to be 97° F. She had suffered a throat infection with vomiting, and had been treated with sulphanilamide. On examination of the patient, her throat was normal, but there was some puffiness around the eyes. A short, soft, apical systolic murmur was heard. She was instructed to be up for one hour per day, and after one week for two hours per day. On January 29 she was again examined; she had been up for three hours per day, and her condition was good. Examination of the heart revealed no abnormality. Instructions were given that she should be allowed up for six hours per day.

Except for a mild attack of bronchitis her progress was uneventful until June 24, when she was attending school each afternoon, and subsequently she was allowed to attend school all day. On July 29 she suffered an attack of tonsillitis, with pain in the legs. She attended the casualty department of the hospital, and was ordered home to bed, where she remained for the succeeding ten days. On examination of the patient at this visit the tonsils were observed to be ragged, but not much enlarged. There was no enlargement of the lymphatic glands of the anterior triangles of the neck. The heart was normal. It was decided that a conservative policy should be observed in regard to the matter of tonsillectomy. At the succeeding visit on August 12 the tonsils were observed to be small and the question of operation was deferred, although the patient had suffered a number of attacks of sore throat.

On November 11 examination of the heart gave negative results, and the patient was attending school all day. She was feeling well, and not becoming unduly tired. Prophylactic sulphanilamide treatment was commenced. She attended again on February 17, 1945, when the heart was found to be normal in size on clinical examination, but a faint apical systolic murmur was heard. The patient had had an attack of mumps two months previously, and at that time prophylactic sulphanilamide treatment had been stopped. The apical systolic murmur was heard again at the next visit on April 14, and sulphanilamide treatment had been resumed five weeks previously.

On May 26 she was reported to have been in bed for seven days, complaining of "stiff legs", which had improved at the time of examination. Examination of the heart gave negative results. She was referred to the out-patient surgical department on account of her turning over on her feet when walking, and subsequently she had "knock knee" splints fitted. She was last examined on December 1, 1945, when she was found to be well, and physical examination revealed no abnormality. Material from throat swabs examined during the course of prophylactic sulphanilamide treatment failed to yield cultures of hæmolytic streptococci.

This case is typical of many seen during the course of my experience of juvenile rheumatism. It illustrates in a general way the plan of treatment of rheumatic fever adopted at the Children's Hospital Melbourne. In my review of 1943, the time spent in hospital was stated to be as follows: under three months, 45·47%; three months to five months, 23·14%; six months to eight months, 10·02%; and over eight months, 21·37%. As was remarked in 1943, the plan of treatment adopted resulted in 35·57% of the children who could be followed up escaping without discoverable cardiac damage. This figure agrees closely with other published statistics. This case also reveals the distinction between "growing pains" and subacute rheumatism. For some reason, at present not fully understood, this child was able to withstand the incidence of infection, and from the course of the disease in her case it seems possible that the use of sulphanilamide and sodium salicylate shortly after, or at the onset of, upper respiratory tract infection, may have played some part in controlling the rheumatic process. On the other hand, she appears to have had natural resistance apart from the effects of prophylactic measures, for the first attack for which she was treated did not result in any evident cardiac impairment.

### Diastolic Murmurs.

The two following cases illustrate apical diastolic murmurs heard over a long period without apparent impairment of cardiac function, or any advance towards the development of mitral stenosis.

CASE V.—E.B., a male child, aged nine years and eleven months, was admitted to the Children's Hospital, Melbourne, on November 4, 1942, complaining of abdominal pain present for the previous four days. Two days prior to his admission to hospital he had pains in his feet and legs and was unable to walk. The day before his admission he complained of pain in his back, and on the day after his admission to the hospital he was unable to straighten his right knee joint and he had pain in the groin and hips. He had had an attack of rheumatic fever at the age of three years and was reported to have had pain and swelling of the ankle joints at that time. He spent a few days resting in bed. Examination on his admission to hospital revealed that the apex beat of the heart was in the fifth left intercostal space two inches from the mid-sternal line, in the nipple line. Soft systolic murmurs were heard at all areas, the maximum being at the mitral area. The rhythm was regular. No joint swellings were present.

Under routine treatment the heart appeared normal on examination on February 27, 1943. However, when the child was examined on October 2, apical systolic and diastolic murmurs were heard at the mitral area. The latter murmur was heard early, in diastole, and was short, faint and localized to a small area internal to the apex beat of the heart. No cardiac enlargement was evident. Fourteen days before this visit the patient had had a cold, but no pain anywhere in his body. He had been attending school

XUM

all day and instructions were given that he should remain at home for rest and come in for examination again in seven days. On October 9 he was well and no further change in his cardiac condition was observed.

On September 2, 1944, he was well and gaining in weight. As before, a faint, apical, systolic murmur was audible and also an early, short, localized, diastolic murmur. He had been forbidden to engage in organized games. When he was last examined, on August 18, 1945, faint systolic murmurs were heard at the aortic and pulmonary areas, and the pulmonary second sound was unduly accentuated. The cardiac signs otherwise were found to be as previously recorded. The apical diastolic murmur remained faint and localized to an area internal to the apex beat, and there was no clinical evidence of cardiac enlargement.

Case VI.—F.R., a male child, aged twelve years and ten months, was admitted to the Children's Hospital, Melbourne, on December 7, 1942, complaining of a sore throat from which he had suffered six weeks previously. Two weeks before his admission to hospital he had had earache, followed by pain and swelling in the knee joints, and he had been thin and listless for the same period. On examination of the patient his temperature was 99·4° F. The apex beat of the heart was in the fifth left intercostal space three inches from the mid-sternal line, in the nipple line. A soft systolic murmur was audible at the mitral area and the pulmonary second sound was accentuated. Sinus arrhythmia was evident. No joint swellings were present.

Table II.

*Variation of Alert and Sleeping Pulse Rates in Rheumatic Infection. (After* Schlesinger, Modified.)

| Diagnosis. | Alert Rate. (Per Minute.) | Sleeping Rate. (Per Minute.) | Difference. |
|---|---|---|---|
| *Age 5 to 10 Years.* | | | |
| Rheumatic heart disease (quiescent) | 88 | 78 | 10 per minute. |
| Mitral regurgitation .. .. | 87 | 77 | 10 per minute. |
| Mitral stenosis .. .. .. | 89 | 78 | 11 per minute. |
| *Age 10 to 15 Years.* | | | |
| Rheumatic heart disease (quiescent) | 84 | 75 | 9 per minute. |
| Mitral regurgitation .. .. | 84 | 74 | 10 per minute. |
| Mitral stenosis .. .. .. | 85 | 75 | 10 per minute. |
| *Active Carditis.* | | | |
| Nodules .. .. .. .. | 100 | 96 | 4 per minute. |
| Arthritis .. .. .. .. | 118 | 112 | 6 per minute. |
| Nodules and chorea .. .. | 110 | 105 | 5 per minute. |
| Cardiac failure, nodules .. .. | 118 | 116 | 2 per minute. |
| Pericarditis, nodules .. .. | 116 | 116 | 0 per minute. |

On December 21 he was transferred to the convalescent hospital where his progress was slow owing to fluctuations in the sleeping pulse rate. On November 6, 1943, routine examination revealed the presence of systolic and diastolic murmurs at the apex of the heart, the former being faint and the latter early, short, faint and localized to a small area internal to the apex beat. He had been well except for a mild attack of scarlet fever two months previously.

On February 17, 1945, having attained the age of fourteen years, the patient was transferred to an adult hospital. Examination at this time revealed faint systolic murmurs at the aortic, pulmonary, tricuspid and mitral areas. The diastolic murmur at the mitral area remained; it was faint and localized as before, and audible only when the patient was in the recumbent position. It was not heard on quickening of the heart rate. There was no clinical evidence of cardiac enlargement.

Case VII.—J.D., a male child, aged eight years and six months, was admitted to the Children's Hospital, Melbourne, on May 12, 1941, complaining of pain in the right ankle joint. Three days previously he had contracted a severe cold. At the age of five years he had had an attack of rheumatic fever, for which he had been confined to bed for a period of five months. His family history was irrelevant except that his father had had "rheumatics". On examination of the patient, his temperature was 101° F. and his pulse rate 130 per minute. The apex beat of the heart was in the fifth left intercostal space four and a half inches from the mid-sternal line outside the nipple line. The rhythm was regular and a blowing systolic murmur conducted towards the axilla was audible. The right ankle joint was painful on movement, but not swollen. Otherwise physical examination revealed no abnormality. He was given routine treatment. On May 27 the apex beat was in the fifth left intercostal space half an inch outside the nipple line. Systolic murmurs were audible at the mitral, pulmonary, aortic and tricuspid areas, and a faint localized diastolic murmur was heard at the mitral area. By January 17, 1942, he was well. No cardiac enlargement was evident on physical examination. A faint apical systolic murmur was audible and no diastolic murmur was heard at the mitral area. On February 14 he was allowed to go to school for half a day on five days of the week.

On November 28, 1942, he was attending school all day, and examination of the heart revealed no abnormality. He remained well, with no change in clinical signs, until August 5, 1944, when it was found that he had a faint apical systolic murmur and a diastolic murmur at the left border of the sternum at and below the second left costal cartilage. The heart was not enlarged.

On December 2, 1944, the cardiac signs were as formerly recorded. The pulse rate was 136 per minute, owing to excitement incidental to physical examination. The systolic blood pressure was 160 millimetres of mercury and the diastolic pressure 60 millimetres. Radiological examination revealed enlargement of the cardiac silhouette. The hæmoglobin value was 87%, and the red cells numbered 5,500,000 per cubic millimetre. There was no change in the character of the pulse, nor any capillary pulsation. The retinal vessels were normal on ophthalmoscopic examination. When last examined in July, 1946, the patient was well. No cardiac enlargement was detected. The apical systolic murmur formerly present was not heard, but a diastolic murmur was again evident at the left border of the sternum, and was conducted downwards to a point opposite the third left intercostal space. Radiological examination, with the œsophagus outlined by barium, was undertaken by Dr. Colin Macdonald, honorary radiologist to the hospital. It revealed no evidence of enlargement of the left auricle of the heart or anything radiologically to suggest mitral stenosis.

This case is most illustrative and of a type rarely encountered. It reveals that, as far as clinical examination shows, although the mitral valve may be affected in acute rheumatism, such affection may be temporary, the main incidence of the rheumatic process falling on the aortic valve. Much has been written on the question of conduction of diastolic murmurs down the left border of the sternum to the region of the mitral area of the heart. I believe that all murmurs heard at the base of the heart, to the left of the sternum, should be carefully "timed" by the finger placed over the carotid artery in the neck. I have been convinced, by personal observation, that diastolic murmurs heard at the base of the heart are difficult to detect in the early stages of aortic disease due to rheumatic infection. I further consider that these murmurs have a quality which, when once heard and recognized, easily differentiates them from diastolic murmurs originating in the mitral area. This quality is one of softness combined with a distinctive resonance. Coombs[75] wrote of these murmurs: "In many cases the bruit is heard right down to the apex." Coombs also described cases in which he observed reduplication of the second sound at the mitral area, and showed how this reduplicated sound changed to a second sound with a faint bruit following the murmur. Subsequently the mitral second sound might be replaced by a murmur. I have observed that in some cases the short localized diastolic murmur at the mitral area is subsequently heard over a wider area around the apex beat, and in such cases radiological examination has revealed increase in size of the left auricle of the heart. These observations lead to the conclusion that short apical diastolic murmurs should be carefully noted from time to time and the extent of the precordium over which they are audible carefully recorded.

### Epidemiology and Control.

Griffith[76] studied hæmolytic streptococci of human origin. He began with a serological investigation of a series of streptococcal cultures obtained in cases of scarlet fever. Griffith found that they were not uniform in sero-

logical characteristics, and that certain types, defined by agglutination reactions, predominated. The dominant strains in a series of 222 strains were four in number: type I, 4·5%; type II, 26·5%; type III, 20·3%; type IV, 19·8%. The remaining 29·7% were a heterogeneous group in which were many serological types. Griffith identified 27 types. Type XVII ("Beatty") was isolated and identified in an outbreak of tonsillitis and *otitis media* followed by acute rheumatism at a public school. Lancefield[77] found that all except two of 106 strains of hæmolytic streptococci isolated from man and animals could be classified into five groups by the use of a precipitin test. Group A comprised largely strains of human origin; group B comprised strains from mastitis in cows, and from normal milk; group C was derived from various lower animals; group D was isolated from cheese, and group E from a few strains from certified milk. Many other groups have since been identified. The group-specific substance present in strains of group A has been identified as carbohydrate in nature. Modern classification embraced both Lancefield's groups and Griffith's types. Thus group A, type II, and type XVII have been isolated in Melbourne. No specific type of hæmolytic streptococcus has been identified as the cause of acute rheumatism, but the type responsible may vary with latitude and climate. The nucleoprotein fraction of the hæmolytic streptococcus is partly common to the various groups, and consequently no specific diagnostic test for acute rheumatism by the use of immunological procedures is available. Six factors have been isolated from products of hæmolytic streptococci —namely, streptolysin O and streptolysin S, leucocydin, fibrinolysin, hyaluronidase, and the erythrogenic toxin responsible for the rash of scarlet fever. The factors streptolysin O and S and fibrinolysin have been utilized by various workers in attempts to elucidate the immunological reactions in rheumatic fever.

Streptococci are regarded essentially as parasites of the pharyngeal mucosa, and their potency as infective agents depends on their capability of multiplying freely in the mucous membrane of the pharynx. The epidemic quality depends on the type of organism, as in scarlet fever, and on the condition of the host. Given suitable conditions in the patient, there will be free and active production of toxin. The result of an epidemic of upper respiratory tract infection may be an outbreak of scarlet fever, with which the erythrogenic toxin of the streptococcus is associated; or acute nephritis may supervene, and this may be essentially an allergic manifestation. Rheumatic fever may become prevalent and is regarded as an allergic response to streptococcal products, but this response cannot be worked out in detail (Burnet).[78] The epidemiological factors responsible are as follows (Burnet): (i) poverty and poor housing conditions, (ii) cold and exposure, (iii) hereditary influences, (iv) the possibility of sensitization to streptococcal products from the age of three years to puberty. The factors in regard to infectivity from person to person of the upper respiratory tract infections believed to precede the rheumatic episode are conveyance of infection by droplets and droplet nuclei from the respiratory passages. The former fall rapidly in the air and come to rest on bedclothes as in a hospital ward, and on the floor. The nuclei remain suspended in air for some time. Control of droplet infection can be effected by using substances of the nature of paraffin oil on floors. The droplet nuclei are controlled only by ultra-violet light installations or by aerosols. Suitable oil can be applied to laundered articles. The subject has recently been fully discussed.[79] The importance of infection, and in particular of diagnosis of the initial attack, has been suggested. Stroud, Goldsmith, Polk and Thorp,[80] after ten years' study, concluded that primary manifestations of rheumatic fever frequently went undetected, and that heart damage discovered years later, might be regarded as due to a first infection. Rosenblum and Rosenblum[81] studied seventy rheumatic families, and suggested that particular attention should be paid to infection in other members of a family. By so doing, they think it may be possible to prevent rheumatic infection in susceptible persons, and to commence treatment early should it occur.

The problems of epidemiology and control have been discussed by Miller,[82] by Poynton[83] and by Glover.[84] Reviews on the incidence of rheumatic infection in New South Wales[85] and in South Australia[86] have been published, and H. Boyd Graham[87] has written on the incidence in Victoria. All agree that further knowledge of the mechanism of the rheumatic process is desirable. Improved living conditions are essential. According to resolutions of the Cardiac Society and the British Pædiatric Association[88] rheumatic infection should be a notifiable disease, provided adequate facilities for diagnosis and treatment are available.

The modern conceptions have been well summarized by Schwentker,[89] who writes as follows: "The epidemiology of streptococcal infections is a summation of those factors concerning the host population and those related to its environment, including the causative organism." Schwentker states that the morbidity of streptococcal disease is related to latitude and season. Only a few types of streptococci are active in a community at any specific time. Some active strains may produce all types of streptococcal disease. Other pathogenic strains have only a single ability to produce scarlet fever. The types present in any community and their pathogenicity vary from year to year. The carrier rate of any strain affects its dissemination and the degree to which the population is affected. Increased power of dissemination by any one strain may result in an epidemic. Some individuals in a population may escape in an epidemic by virtue of some protective mechanism associated with immunity, although exposed to heavy infection. This immunity may be type-specific.

The London County Council's rheumatism scheme[90] is claimed to have decreased the incidence of rheumatic heart disease in London school children from 2·2% to 0·77% since its inception. This scheme embraces supervisory centres, the functions of which are summarized as being supervisory, diagnostic, educational and social. Certain centres are organized in association with the out-patient departments of hospitals. Independent centres have facilities for examination of patients and investigational procedures such as hæmoglobin estimations and blood sedimentation rate determinations, the latter being regarded as essential. Physicians in charge of the centres are of consultant rank, and the number of patients examined at any one session is strictly limited. Social workers are regarded as important personnel in these centres.

The London County Council units have been useful sources of essential statistical data. Thus, in 1,000 consecutive cases admitted to the rheumatic units of the London County Council in 1937, the distribution of the types of attack was as follows: acute polyarthritis, 185 (18·5%); subacute rheumatism, 520 (52·0%); chorea, 190 (19·0%); chorea and concurrent subacute rheumatism, 105 (10·5%). In this statement cases of insidious carditis are included in the subacute rheumatism group. In an analysis of 3,477 case records of children attending rheumatic supervisory centres, 53% revealed a history of subacute rheumatism.

In a rheumatic unit associated with a general hospital the in-patient and out-patient physicians may correspond to the physicians of consultant rank attached to supervisory centres. If a rheumatic clinic is attached to the hospital, as is the case at the Children's Hospital, Melbourne, this clinic can act as a consultant unit in association with in-patient and out-patient physicians.

### Prophylaxis.

Prophylaxis by penicillin may prove to be useful in controlling the possible spread of sulphanilamide-resistant streptococci in those patients with developing mitral stenosis, established mitral stenosis, and mitral and aortic involvement in the rheumatic process, and the occasional patient with pure aortic valve infection, who have been treated prophylactically with one of the sulphonamides. Knott and Blaikely[91] have used penicillin sprays to counteract the spread of upper respiratory tract infections, and their results show that a 50% improvement has been obtained. As penicillin, like the sulphonamides, is a bacteriostatic agent only, as a therapeutic measure it has no effect on the rheumatic process. Penicillin has been administered in rheumatic infection at the Children's Hospital, Melbourne, and found to be of no value. This

observation has been supported by Watson, Rothbard and Swift,[91] and by others.[92]

Coburn and Moore[93] have investigated the possibility of prophylaxis by the use of salicylate of sodium during the first and second phases of the acute rheumatic attack and believe that it has been of benefit in preventing the inset of rheumatic fever. Coburn and Moore claim that statistical treatment of their data indicates that the association between prophylaxis by sodium salicylate and the failure of rheumatic subjects to have attacks after streptococcal pharyngitis is significant. It has been shown that the administration of sodium bicarbonate along with sodium salicylate lowers the concentration of salicylate in the blood serum. The use of enteric-coated capsules overcomes the tendency to nausea and vomiting frequently incidental to the use of sodium salicylate without the addition of sodium bicarbonate. Wegria and Smull[94] conclude that the course of acute rheumatic fever may possibly be shortened if "adequate" salicylate therapy is commenced within a week or two of the onset. Acetyl salicylic acid has been advocated[95] to replace sodium salicylate in the treatment of rheumatic fever, and it is stated that this drug may be given in large doses even to small children, if it is combined with glucose.

When the last review was made in 1943, a full statement of the position of sulphanilamide prophylaxis was outlined. Since then there have been other papers,[85][86][87][88] including one investigation at the Children's Hospital, Melbourne. All have shown the efficacy of sulphonamide drugs in preventing "recurrences", but Wilson and Lubschez's survey[18] proved the results invalid in most of the studies. More recently this procedure has been tried in American military and naval establishments, and has failed to prove its value as a general prophylactic measure.[100] In these cases, the increase in or recurrence of high incidence of upper respiratory tract infections, of scarlet fever and of other infections has been shown to be attributable to influx of new susceptible recruits.[101] All reviews in the rheumatic fever series have been made on subjects who have suffered at least one attack of acute rheumatism. Therefore, as was mentioned in 1943, it is possible that the "mechanism" of the "recurrence" in acute rheumatism may be different from that of the initial attack.

Sulphadiazine has been much used,[102] because it is less likely to cause complications than other sulphonamide drugs, and because its level is more easily maintained in the blood. The blood concentration of the sulphonamides when used prophylactically is subinhibitory, being of a maximum level of two to three milligrammes *per centum*. The risk of appearance of resistant strains of streptococci is run; this has occurred in the material obtained from the Children's Hospital, Melbourne, where prophylaxis is undertaken. The risk is not inconsiderable, especially where poor living conditions with overcrowding are prevalent. Siegel and Julianelle[103] studied the effect of prolonged administration of sulphadiazine prophylactically in relation to the development of resistant strains of pneumococci in normal children. They found that some types may acquire a high degree of resistance. Despite their fastness, these strains retained their virulence and specificity. Sigel and Julianelle used controls, and found that precipitating, agglutinating and protective antibodies were not found in either treated or untreated children.

Wasson and Brown[104] claim to have had success in preventing recrudescences by injections of graduated doses of hæmolytic streptococcus filtrate.

### Convalescence.

Provision is made overseas for special training during convalescence—namely, games, walks and school.[104] Much is done here to give advice and help in future occupation for those patients who unfortunately have seriously damaged hearts; advice is also given, and frequently much-needed assurance, to those who can and should lead a normal life, free from anxiety about their cardiac condition. In this also the almoner department is most helpful. No special exercise training has been possible in Melbourne, but Sherbrooke and Frankston convalescent establishments are registered schools. Occupational therapy now extends to the home, and has been particularly useful for patients suffering from hemichorea, after they leave the convalescent hospital. The almoner department is essential in this scheme.

### Summary.

1. A review is presented of juvenile rheumatism as seen at the Children's Hospital, Melbourne, during the years 1942 to 1945.

2. Emphasis is laid on the diagnosis of insidious cases.

3. The ætiology and pathology of rheumatic infection are briefly considered.

4. The early diagnosis and management of rheumatic fever are discussed.

5. Reference is made to investigational methods.

6. The importance of diastolic murmurs is considered.

7. Epidemiology and control of streptococcal infections are outlined.

8. Prophylactic measures are mentioned.

### Acknowledgements.

I wish to thank members of the honorary medical staff of the Children's Hospital, Melbourne, for permission to investigate patients under their care. My thanks are due particularly to Dr. J. W. Grieve for his constant help, advice and encouragement during the course of this study, and for his unfailing interest in the progress of the work. Dr. H. Lawrence Stokes has kindly allowed me free access to the patients in the rheumatic clinic. Dr. H. Boyd Graham, who first directed the rheumatic clinic at the hospital, has been most helpful. Dr. Colin Macdonald, honorary radiologist at the hospital, has kindly discussed with me the radiological findings in one very instructive case. My thanks are also due to Dr. J. H. Bolton, honorary out-patient physician at the Royal Melbourne Hospital, for permission to refer to one of his patients. The almoner to the hospital, Miss Hodge, and her staff, have been most helpful at all times.

### References.

(1) G. E. M. Scott: "Rheumatic Infection in Childhood: A Survey from the Children's Hospital, Melbourne", THE MEDICAL JOURNAL OF AUSTRALIA, October 16, 1943, page 309.

(2) A. R. Coburn: "The Factor of Infection in the Rheumatic State", 1931.

(3) G. Hadfield and L. P. Garrod: "Recent Advances in Pathology", 1938.

(4) A. R. Rich and M. R. Lewis: "The Nature of Allergy in Tuberculosis as Revealed by Tissue Culture Studies", *Bulletin of the Johns Hopkins Hospital*, Volume L, 1932, page 115.

(5) J. D. Aronson: "The Specific Cytotoxic Action of Tuberculin in Tissue Culture", *The Journal of Experimental Medicine*, Volume LIV, 1931, page 387.

(6) E. L. Opie: "The Pathogenesis and Transmission of Tuberculosis", *Acta medica scandinavica*, Volume CLXXIX, 1930, page 104.

(7) A. K. Krause: "Studies on Tuberculous Infection. XII. The Dissemination of Tubercle Bacilli in the Immune Guinea Pig, with a Discussion of Probable Factors Involved in Tuberculo-immunity", *American Review of Tuberculosis*, Volume XIV, 1926, page 211; quoted by Hadfield and Garrod, *loco citato*.

(8) H. S. Willis: "Studies on Tuberculous Infection. X. The Early Dissemination of Tubercle Bacilli after Intracutaneous Inoculation of Guinea Pigs of First Infection", *American Review of Tuberculosis*, Volume XI, 1925, page 439; quoted by Hadfield and Garrod, *loco citato*.

(9) A. R. Rich: "The Role of Allergy in Tuberculosis", *Archives of Internal Medicine*, Volume XLIII, 1929, page 691.

(10) A. R. Rich and H. A. McCordock: "An Enquiry Concerning the Role of Allergy, Immunity and other Factors of Importance in the Pathogenesis of Tuberculosis", *Bulletin of the Johns Hopkins Hospital*, Volume XLIV, 1929, page 273.

(11) W. R. F. Collis, W. Sheldon and N. Gray Hill: "Cutaneous Reactions in Acute Rheumatism", *The Quarterly Journal of Medicine*, Volume XXV, 1932, page 511.

(12) H. J. Gibson, W. A. R. Thomson and D. Stewart: "The Hæmolytic Streptococcus as a Factor in the Causation of Acute Rheumatism", *Archives of Disease in Childhood*, Volume VIII, 1933, page 57.

(13) L. M. Taran, J. M. Jablon and H. N. Weyr: "Immunologic Studies in Rheumatic Fever", *The Journal of Immunology*, Volume XLIX, 1944, page 209.

(14) A. F. Coburn and R. H. Pauli: "The Significance of Prolonged Streptococcal Antibody Development in Rheumatic Fever", *The Journal of Clinical Investigation*, Volume XVIII, 1939, page 141.

(15) L. A. Rantz, W. M. M. Kirkby and A. H. Jacobs: "Group A Hæmolytic Streptococcus Antibodies, Griffith Type Agglutinin and Antistreptolysin Titres in Normal Men and in Acute Infections", *The Journal of Clinical Investigation*, Volume XXII, 1943, page 411.

(17) M. G. Wilson and R. Lubschez: "Recurrence Rates in Rheumatic Fever", *The Journal of the American Medical Association*, Volume CXXVI, 1944, page 477.
(17) E. F. Bland and T. D. Jones: "The Delayed Appearance of Heart Disease after Rheumatic Fever", *The Journal of the American Medical Association*, Volume CXIII, 1939, page 1380.
(18) Professor Aschoff: *British Medical Journal*, Volume II, 1906, page 1103.
(19) W. C. von Glahn and A. M. Pappenheimer: "Specific Lesions of Peripheral Blood Vessels in Rheumatism", *The American Journal of Pathology*, Volume II, 1926, page 1926.
(20) J. J. Robinson: "Notes on Some Experimental Streptococcal Injury in the Rabbit and Guinea Pig", *Archives of Pediatrics*, Volume XLI, 1944, page 564.
(21) A. R. Rich and J. E. Gregory: "Further Experimental Cardiac Lesions of the Rheumatic Type Produced by Anaphylactic Hypersensitivity", *Bulletin of the Johns Hopkins Hospital*, Volume LXXV, 1944, page 115.
(22) A. R. Rich and R. H. Follis: "Studies on the Site of Sensitivity in the Arthus Phenomenon", *Bulletin of the Johns Hopkins Hospital*, Volume LXVI, 1940, page 106.
(23) A. R. Rich: "The Significance of Hypersensitivity in Infections", *Physiological Reviews*, Volume XXI, 1941, page 70.
(24) H. Selye and E. I. Pentz: "Pathological Correlations Between Periarteritis Nodosa, Renal Hypertension and Rheumatic Lesions", *The Canadian Medical Association Journal*, Volume XLIX, 1943, page 264.
(25) C. C. Smith, P. M. Zeak and J. McGuire: "Periarteritis Nodosa in Experimental Hypersensitive Rats and Dogs", *The American Journal of Pathology*, Volume XX, 1944, page 721.
(26) C. A. Green: "Researches into Ætiology of Acute Rheumatism; Rheumatic Carditis; Post-Mortem Investigation of Nine Consecutive Cases", *Annals of Rheumatic Diseases*, Volume I, 1939, page 86; quoted by Collis, *loco citato*.
(27) W. R. F. Collis: "Bacteriology of Rheumatic Fever", *The Lancet*, Volume II, 1939, page 817.
(28) S. Thompson and J. Innes: "Hæmolytic Streptococci in the Cardiac Lesions of Acute Rheumatism", *British Medical Journal*, Volume II, 1940, page 733.
(29) R. J. Wright-Smith: "The Pathology of Rheumatic Carditis", *Royal Melbourne Hospital Clinical Reports*, Volume XVI, 1945, page 13.
(30) W. B. Cheadle: "Clinical Lecture on an Outbreak of Rheumatic Pneumonia", *The Lancet*, Volume I, 1888, page 861.
(31) A. E. Naish: "The Rheumatic Lung", *The Lancet*, Volume II, 1928, page 10.
(32) A. D. Fraser: "The Aschoff Nodule in Rheumatic Pneumonia", *The Lancet*, Volume I, 1930, page 70.
(33) L. Finlay: "Pulmonary Lesions in Rheumatism", *Archives of Disease in Childhood*, Volume V, 1930, page 259.
(34) A. F. Coburn: "Relationship of the Rheumatic Process to the Development of Alterations in Tissues", *American Journal of Diseases of Children*, Volume XLV, 1933, page 933.
(35) M. A. Rabinowitz: "Rheumatic Pneumonia", *The Journal of the American Medical Association*, Volume LXXXVII, 1926, page 142.
(36) G. Hadfield: "The Rheumatic Lung", *The Lancet*, Volume II, 1938, page 710.
(37) E. Z. Epstein and H. B. Greenspan: "Rheumatic Pneumonia", *Archives of Internal Medicine*, Volume LXVIII, 1941, page 1074.
(38) R. Rich and J. E. Gregory: "On the Anaphylactic Nature of Rheumatic Pneumonia", *Bulletin of the Johns Hopkins Hospital*, Volume LXXIII, 1943, page 465.
(39) K. F. Neuburger, E. F. Greever and E. K. Rutledge: "Rheumatic Pneumonia", *Archives of Pathology*, Volume XXXVII, 1944, page 1.
(40) E. V. Keogh, I. Macdonald, J. Battle, R. T. Simmons and S. Williams: "The Serological Groups and Types of Streptococci Isolated from Excised Tonsils", *British Medical Journal*, Volume II, 1939, page 1036.
(41) S. D. Elliot: "Bacteriæmia Following Tonsillectomy", *British Medical Journal*, Volume II, 1939, page 589.
(42) I. Macdonald, R. T. Simmons and E. V. Keogh: "Further Observations on the Incidence of Hæmolytic Streptococci in the Tonsils", THE MEDICAL JOURNAL OF AUSTRALIA, June 22, 1940, page 849.
(43) A. Castiglioni: "A History of Medicine", 1941.
(44) M. J. Shapiro: "Differential Diagnosis of Nonrheumatic Growing Pains and Subacute Rheumatism", *Journal of Pediatrics*, Volume XIV, 1939, page 315.
(45) J. C. Hawksley: "The Nature of Growing Pains and their Relation to Rheumatism in Children and Adolescents", *British Medical Journal*, Volume I, 1939, page 155.
(46) G. F. Still: "Common Disorders and Diseases of Childhood", Fifth Edition, 1927.
(47) A. Jacobi: *Archives of Pediatrics*, Volume VII, 1889, page 353; quoted by Shapiro, *loco citato*.
(48) J. W. Grieve: Personal communication.
(49) S. Gibson: "The Clinical Significance of Heart Murmurs in Children", *Medical Clinics of North America*, Pediatrics, January, 1946, page 35.
(50) W. T. Ritchie: "Acute Rheumatic Carditis", *British Medical Journal*, Volume II, 1939, page 581.
(51) C. F. Coombs: "The Diagnosis and Treatment of Rheumatic Heart Disease in its Early Stages", *British Medical Journal*, Volume I, 1930, page 227.
(52) J. Mackenzie: "Diseases of the Heart", Fourth Edition, 1925.
(53) R. Fahreus: "The Suspension Stability of the Blood", *Acta medica scandinavica*, Volume LV, 1921, page 1.
(54) A. Westergren: "Studies of the Suspension Stability of the Blood in Pulmonary Tuberculosis", *Acta medica scandinavica*, Volume LIV, 1920-1921, page 247.
(55) A. Carlton Ernstein: "Erythrocyte Sedimentation, Plasma Fibrinogen and Leucocytosis as Indices of Rheumatic Infection", *The American Journal of the Medical Sciences*, Volume CLXXX, 1930, page 12.
(56) I. T. Zeckwer and Helen Goodell: "The Sedimentation Rate of Erythrocytes", *The American Journal of the Medical Sciences*, Volume CLXIX, 1925, page 209.
(57) M. G. Peterman and S. J. Seeger: "Sedimentation Reaction in Children", *American Journal of Diseases of Children*, Volume XXXVII, 1929, page 693.
(58) A. Landau: "Microsedimentation (Linzenmeier-Raunert Method)", *American Journal of Diseases of Children*, Volume XLV, 1933, page 691.
(59) M. M. Wintrobe: "Macroscopic Examination of the Blood", *The American Journal of the Medical Sciences*, Volume CLXXXV, 1933, page 58.
(60) G. E. Beaumont and J. W. Maycock: "The Erythrocyte Sedimentation Rate", *The Lancet*, Volume II, 1935, page 19.
(61) M. Hynes and L. E. H. Whitby: "Correction of the Sedimentation Rate for Anæmia", *The Lancet*, Volume II, 1938, page 249.
(62) W. W. Payne: "Acute Rheumatism and the Sedimentation Rate", *The Lancet*, Volume I, 1932, page 74.
(63) J. W. Cutler: "The Practical Application of the Blood Sedimentation Test in General Medicine", *The American Journal of the Medical Sciences*, Volume CLXXXIII, 1932, page 643.
(64) F. Bach and N. Gray Hill: "Erythrocyte Sedimentation Rate in Rheumatic Fever", *The Lancet*, Volume I, 1932, page 75.
(65) R. R. Struthers and H. L. Bacal: "Determination of the Activity of Rheumatic Infection in Childhood", *The Canadian Medical Association Journal*, Volume XXIX, 1933, page 470.
(66) C. Bruce Perry: "The Sedimentation Rate in Rheumatic Carditis", *Archives of Disease in Childhood*, Volume IX, 1934, page 285.
(67) W. W. Payne and B. Schlesinger: "A Study of the Sedimentation Rate in Juvenile Rheumatism", *Archives of Disease in Childhood*, Volume X, 1935, page 403.
(68) B. Schlesinger: "A Study of the Sleeping Pulse Rate in Rheumatic Children", *The Quarterly Journal of Medicine*, Volume XXV, 1932, page 67.
(69) E. E. Osgood and R. L. Baker: "Erythrocyte, Hæmoglobin, Cell Volume and Saturation Index Standards for Normal Children of School Age", *American Journal of Diseases of Children*, Volume L, 1935, page 343.
(70) H. M. M. MacKay, L. Wills and K. Bingham: "Economic Status and the Hæmoglobin Level of Children of Men in the Fighting Forces and of Civilians", *British Medical Journal*, Volume I, 1946, page 711.
(71) W. Sheldon: "Diseases of Infancy and Childhood", Fourth Edition, 1943.
(72) H. W. Elghammer: "Rheumatic Fever Diagnostic Criteria and Rheumatic Heart Disease", *Medical Clinics of North America*, Pediatrics, January, 1946, page 25.
(73) H. E. B. Pardee: "Clinical Aspects of the Electrocardiogram", 1924.
(74) W. Evans: "A Student's Handbook of Clinical Electrocardiography", 1934.
(75) C. F. Coombs: "Rheumatic Heart Disease", 1924.
(76) F. Griffith: "The Serological Classification of Streptococcus Pyogenes", *The Journal of Hygiene*, Volume XXXIV, 1934, page 542.
(77) R. C. Lancefield: "The Serological Differentiation of Human and Other Groups of Hæmolytic Streptococci", *The Journal of Experimental Medicine*, Volume LVII, 1933, page 571.
(78) F. M. Burnet: Melbourne Permanent Post-Graduate Committee Lectures, 1945 (unpublished).
(79) A. G. Watkins *et alii*: "Cross Infection in Children's Wards; A Report Presented to the British Pædiatric Association", *British Medical Journal*, Volume I, 1946, page 673.
(80) W. D. Stroud, M. A. Goldsmith, D. Stewart Polk and F. Q. Thorp: "Ten Years' Observation of Children with Rheumatic Heart Disease", *The Journal of the American Medical Association*, Volume CI, 1933, page 502.
(81) A. Rosenblum and R. L. Rosenblum: "A Study of Seventy Rheumatic Families", *American Heart Journal*, Volume XXIII, 1942, page 71.
(82) R. Miller: Discussion on "The Ætiology and Treatment of Heart Disease in Early Life", British Medical Association annual meeting, 1923, *British Medical Journal*, Volume II, 1923, page 704.
(83) F. J. Poynton: "Prevention of Acute Rheumatism", *British Medical Journal*, Volume II, 1924, page 986.
(84) J. A. Glover: "Milroy Lectures on the Incidence of Rheumatic Diseases", *The Lancet*, Volume I, 1930, page 499.
(85) J. K. Maddox: "Metropolitan and Rural Incidence and Distribution of Rheumatic Heart Disease in New South Wales", THE MEDICAL JOURNAL OF AUSTRALIA, March 13, 1937, page 394.
(86) "Report on the Epidemiology of Rheumatic Infection in South Australia", THE MEDICAL JOURNAL OF AUSTRALIA, April 6, 1940, page 461.
(87) H. Boyd Graham: "The Incidence of Rheumatic Infections in Victoria", THE MEDICAL JOURNAL OF AUSTRALIA, June 19, 1937, page 944.
(88) C. E. Thornton: "Child Health: The Campaign Against Juvenile Rheumatism", *The Practitioner*, January-June, 1945, page 112.
(89) F. Schwentker: "The Epidemiology of Streptococcal Infections", *The Journal of Bacteriology*, Volume XLVII, 1944, page 405.
(90) F. A. Knott and J. B. Blaikley: "Penicillin Sprays and Upper Respiratory Tract Infections", *British Medical Journal*, Volume I, 1946, page 349.

(91) R. F. Watson, S. Rothbard and H. F. Swift: "The Use of Penicillin in Rheumatic Fever", *The Journal of the American Medical Association*, Volume CXXVI, 1944, page 274.
(92) F. P. Foster, G. C. McEachern, J. H. Miller, F. E. Ball, C. S. Higley and H. A. Warren: "The Treatment of Rheumatic Fever with Penicillin", *The Journal of the American Medical Association*, Volume CXXVI, 1944, page 281.
(93) A. F. Coburn and L. V. Moore: "Salicylate Prophylaxis in Rheumatic Fever", *The Journal of Pediatrics*, Volume XXI, 1942, page 180.
(94) R. Wegria and K. Smull: "Salicylate Therapy in Acute Rheumatic Fever", *The Journal of Pediatrics*, Volume XXVI, 1945, page 211.
(95) A. G. Kuttner and G. Reyersbach: "The Prevention of Streptococcal Upper Respiratory Infections and Rheumatic Recurrences in Rheumatic Children by the Prophylactic Use of Sulfanilamide", *The Journal of Clinical Investigation*, Volume XX, 1943, page 77.
(96) C. A. Chandler and H. B. Taussig: "Sulfanilamide as a Prophylactic Agent in Rheumatic Fever", *Bulletin of the Johns Hopkins Hospital*, Volume LXXII, 1943, page 42.
(97) K. G. Dodge, J. S. Baldwin and M. W. Weber: "The Prophylactic Use of Sulfanilamide in Children with Inactive Rheumatic Fever", *The Journal of Pediatrics*, Volume XXIV, 1944, page 483.
(98) R. H. Feidt: "Sulphanilamide as a Prophylactic Measure in Recurrent Rheumatic Infection", *The American Journal of the Medical Sciences*, Volume CCVII, 1944, page 483.
(99) S. G. Anderson: "The Prophylactic Use of Sulphanilamide in Rheumatic Fever: Preliminary Report", THE MEDICAL JOURNAL OF AUSTRALIA, June 23, 1945, page 635.
(100) Epidemiology Unit Number 22: "Sulphadiazine Resistant Strains of Beta Hæmolytic Streptococci", *The Journal of the American Medical Association*, Volume CXXIX, 1945, page 921.
(101) S. M. Wheeler and T. Duckett Jones: "Factors in the Control of the Spread of Acute Respiratory Infections with Reference to Streptococcal Illness and Acute Rheumatic Fever", *The American Journal of the Medical Sciences*", Volume CCIX, 1945, page 58.
(102) A. F. Coburn: "The Prevention of Respiratory Tract Bacterial Infections in the United States Navy", *The Journal of the American Medical Association*, Volume CXXVI, 1944, page 88.
(103) M. Siegel and L. A. Julianelle: "The Epidemiology of Acute Respiratory Infections Conditioned by Sulfonamides", *Annals of Internal Medicine*, Volume XXII, 1945, page 1.
(104) F. J. Poynton and R. E. Schlesinger: "Recent Advances in the Study of Rheumatism", Second Edition, 1937.
(105) V. P. Wasson and E. E. Brown: "Further Studies in Immunisation Against Rheumatic Fever", *American Heart Journal*, Volume XXIII, 1942, page 291.

### Addendum.

*Social Care of Children with Rheumatic Fever or Chorea.*

All patients with chorea and rheumatic fever are followed up by the almoner. Each home is visited; a social history is then attached to the rheumatic history, and a special note is made of any social difficulties likely to prevent the child from having a reasonably satisfactory convalescence. If necessary from the medical or social point of view, the child is kept in one of our convalescent homes until he or she is able to lead a reasonably active life or until the home conditions are more satisfactory. Unfortunately the number of beds available for convalescence has been restricted during the war, but this situation is improving slowly.

If a child fails to report at the out-patient department as requested, he is followed up by letter; if he still fails to attend, another home visit is paid.

School lessons commence in hospital as soon as the child is well enough, and are continued in the convalescent homes, and when the child goes home he is enrolled with the correspondence school. An attempt is made to see that the child does not fall too far behind the normal grade for his age, and when necessary a voluntary helper is provided to coach the child.

Handcraft tuition is commenced in hospital and continued in the convalescent homes, and a craft worker visits the child in his home to endeavour to keep him supplied with suitable interesting occupation.

When the child reports to the out-patient department, he may borrow books from the lending library, exchange stamps at the stamp exchange, and if desired, see the craft worker for further tuition. An attempt is made to help the child to lead as full and interesting a life as possible during the convalescent period.

When it is decided to transfer a patient to an adult hospital, a social history as well as a medical history is forwarded from this hospital, and thus continuity of supervision is maintained.

## FIBROSITIS AND THE COMMON PAINS OF DAILY PRACTICE.[1]

By MICHAEL KELLY,
*Department of Anatomy and Histology, University of Melbourne.*

Wandering pains are near akin to rheumatism, but may be distinguished from it by their being accompanied neither with swelling nor any discolouring of the skin. Are they not chiefly suffered by those, whose muscular fibres have been weakened, strained or stiffened by long illnesses, profuse bleedings, bruises, irregular living, hard working or the advances of age?

WILLIAM HEBERDEN: Commentaries on the History and Cure of Diseases.

To different people, fibrositis bears varying degrees of reality. It has been a subject for controversy ever since Sir William Gowers suggested the term in 1904; the suggestion itself aroused a good deal of opposition at the time. "Where is there any benefit in morbid anatomy", asked William Bruce (1904), "for burthening pathology with such an outlandish term as 'fibrositis'?" But the idea caught on. Just such a term was needed by Professor Ralph Stockman (1904), who was finishing the first stage of his labours on the pathology of chronic non-articular rheumatism. For years the views of Stockman held sway, but the doubters were merely biding their time. The concept of fibrositis never was firmly established; and in recent years the pendulum has swung in the direction of non-acceptance. An editorial in the *British Medical Journal* (1941) asked: "How long will both these terms—myositis and fibrositis—have to remain names entirely devoid of pathological significance?" *The Lancet* (1942) has expressed the view that fibrositis is of vague pathological and clinical definition, for some sceptics deny its existence; and so it is not surprising that the diagnosis has fallen into disrepute. In the United States, according to Traeger (1937), a very large proportion of physicians deny the existence of fibrositis.

Quite recently, however, a halt has been called to this agnostic tendency; a spirit of inquiry has gone abroad, and many practitioners have shown a disposition to examine the evidence from a judicial point of view. The subject is an important one, having relation to a large proportion of the patients seen in practice; and a great deal of suffering is involved. A large number of patients consult their medical advisers because of somatic pain, and only a relatively small proportion of these present unmistakable physical signs of disease. The first task of this inquiry is to provide an answer to the question: Does fibrositis exist?

### Does Fibrositis Exist?

Few indeed will be found to deny that fibrositis does signify a definite deviation from the normal. Not many will be found to agree with J. L. Halliday (1942) when he wrote: "The theory of fibrositis in its full form clearly corresponds to a mode of obsessional thinking which goes on operating without reference to reality." There can be little doubt that patients do suffer from vague somatic pains; but their physiological and pathological basis remains the subject of debate. The term "fibrositis" was applied by Gowers to a definite pathological change which was known to accompany muscular rheumatism. Rheumatic changes having been observed in some cases, their existence was presumed in many others. Gradually it came to be realized, however, that demonstrable pathological changes were not a common feature of backache and other muscular pains. Fibrositis as a well-defined structural alteration of tissue came to lose a good deal of its significance. But the rheumatic pains have continued to plague the patients and the doctors; a good deal of evidence, moreover, suggests that the pains are accompanied by a physiological change in the fibrous tissues, which proceeds to structural change in only the more advanced cases. The very presence of pain means that

[1] Financial assistance from the National Health and Medical Research Council of Australia is gratefully acknowledged.

there must be a deviation from the normal; some disorder of cells is present which may not be recognizable by the technique of histology. For this presumed disorder of cells, which manifests itself as pain, it is suggested that the term fibrositis be retained.

Most of those who object to the use of the term do not deny the reality of somatic pain, but they are not certain that in the ill-defined group of disorders there is pathological uniformity sufficient to merit a single name. The causes are many and varied (and commonly unknown), and the clinical picture varies from case to case. Fibrositis has the same reality as has rheumatism, whose unpleasant reality cannot be denied, though its pathological basis remains obscure. The use of alternative terms, however, does not bring any nearer a solution of the problem. If we consent to speak of fibrositis, let us not be led thereby into any premature assumptions as to its nature. If we assert that fibrositis is real, we must hasten to add that it signifies a symptom-complex whose cause is unknown, rather than a well-defined disease.

### Is Fibrositis a Specific Disease?

The question "Is fibrositis a specific disease?" could lead to another question: "What is meant by 'a specific disease'?" Though we are taught to divide up disease into a number of specific "morbid entities", those which we can so distinguish form but a small part of the whole body of disease. For a disease to be truly an entity, it must have a well-defined clinical picture, and it must have a single recognizable cause. For present purposes disease can be divided roughly into four categories:

1. Well-defined cause and well-defined clinical picture. The infectious diseases and those directly due to injury belong to this group, with perhaps a few others.
2. Cause unknown, but clinical picture well-defined. Malignant disease, diabetes, peptic ulcer, and many others, including most of the surgical diseases, fall into this category.
3. Those affections which claim neither a constant clinical picture nor a known causation. Here belong most of the ills the flesh is heir to: the rashes, pains and aches, the innumerable deviations from normal which bring humanity flocking to the consulting rooms. Urticaria, for example, is a reaction in the skin which can be recognized as such, but it appears in response to a variety of agents, or to unknown causes. The rheumatic diseases belong to this group.
4. The degenerations which result from senescence or attrition. These really are not diseases at all. With use and increasing years the tissues undergo changes which, if symptomless, are regarded as normal. Sometimes the degenerative changes appear early in life, owing to unknown factors in which heredity may play a part. Premature baldness is not a disease; but early degenerative change in the joints is a disease and is dignified by a name, osteoarthritis.

The habit of thinking of illness in terms of diseases has some undesirable effects. "The student is taught to believe that a 'disease' is a concrete agent that 'does things' and produces symptoms", wrote F. M. R. Walshe (1944). "So taught, he is ill-equipped to deal with new and unfamiliar reactions, and cannot handle new and strange experiences as he should." One result is the state of mind which demands a single cause for each disease. The causes of certain defined entities are known. Therefore (the reasoning appears to proceed) every other disease, such as cancer or rheumatism, must have a single cause which can be found. It is more likely, however, that rheumatism will prove to be due not to a single cause but to the combined action of many factors, which vary from case to case. Fibrositis is a word used to describe a certain kind of tissue reaction which may result from a variety of causes. "It is not a disease", said Poynton and Schlesinger (1937), "but a pathological process underlying various rheumatic disorders, both of a chronic and an acute nature."

### The Relation of Fibrositis to Rheumatic Disease in General.

With such a varied pathological opportunity, the clinical picture of fibrositis may range from a mild or transient localized ache to severe and intractable pain, widely spread and of a crippling nature. Non-articular rheumatism comprises the vast bulk of rheumatic disease—75% at the arthritis clinics in the United States of America, and therefore a much greater proportion in private practice. It is customary in classifications of rheumatic disease to separate non-articular from articular rheumatism. This separation may be useful for purposes of description, but its utility breaks down when it is realized that fibrositic lesions almost invariably accompany every form of arthritis. Collins (1938) and Copeman (1944) hold that fibrositic lesions comprise the main part of the syndrome of rheumatic fever. Stockman (1928) and others have observed many such lesions in every case of rheumatoid arthritis. Gordon (1935) held that the pain in osteoarthritis was due to the accompanying fibrositic lesions, and not to the mechanical deformities, which may occur without pain. It is a grievous error, therefore, to think of fibrositis as a disease which can be separated off from the rest of the rheumatic diseases; more likely is it the basic lesion which occurs in all varieties of rheumatism.

The study of the pyogenic or the tuberculous infections became fruitful only when the significance of the early lesions had been appreciated, and their relation to gross established disease had been traced. Then only did it become evident that previous workers had been in error in attempting to glean all their knowledge from the long-established or generalized infections. The analogy applies to rheumatic disease, whose problem in many quarters is regarded chiefly or wholly as a problem of arthritis. There is much evidence, however, to suggest that fibrositis is the essential lesion in all types of rheumatism. If fibrositis is merely a name for a reaction in the tissues, there is no reason why the name should be withheld from the soft-tissue lesions of rheumatic fever or rheumatoid arthritis. Some cases of rheumatic fever amount to no more than multiple fibrositis; but there is a general hesitancy to make use of the term. In a recent series of cases of rheumatic fever, Copeman many times observed the spread of the process from near-by fibrositic lesions to involve the synovial membrane of a joint. Stockman (1920) and Cassidy (1923) have contended that the arthritic lesions of rheumatoid arthritis are merely examples of chronic fibrositis manifesting itself in joints.

Rheumatism of a joint, therefore, is merely "fibrositis with a difference"; special effects are produced in joints only because of the special characters of the joint tissues.

### The Pathological Changes in Fibrositis.

Acute rheumatism is characterized by the submiliary nodule, a lesion which presents a constant histological appearance (Swift, 1936; Poynton and Schlesinger; Boyd, 1940). When the lesion heals, a patch of fibrosis is left; but in many persistent cases the healing process is incomplete, the cellular and the fibrotic reactions proceeding side by side. As a rule the fibrotic phase is the more prominent. A vicious circle is set up, for the formation of new fibrous tissue means only that additional tissue is present which is susceptible to attack by the rheumatic process. Not only does chronic rheumatism seem to have the power of hastening the degeneration normally brought on by age; but it itself is more especially a disorder of advancing years. The degeneration of age appears to render the tissues more liable to attack by the unknown noxious agent.

The oft-mentioned "nodule" of fibrositis has no relation either to the submiliary nodule or to the larger subcutaneous nodule which sometimes appears in severe generalized rheumatism. The thickenings which Stockman described were in the substance of muscle or fibrous tissues. Various forms of subcutaneous and intramuscular indurations can be found in normal subjects, and doubt has been thrown recently on their significance in fibrositis (Pugh and Christie, 1945). The characteristic lesion of fibrositis manifests itself as a well-defined tender spot; sometimes this is associated with a palpable induration, but more frequently not.

Hench and his collaborators (1941*a* and *b*) have complained more than once of the dearth of original histological investigation of fibrositis. Nearly all of those who have written upon the subject, they say, merely copied the

observations of Stockman; while, of those who have attempted to make original studies, the work has been too sketchy to be of much value.

A review of the literature, however, reveals that a good deal of work has been reported. But, if the results appear to be contradictory, this is because of the nature of the disorder. Collins (1940) removed indurated nodules and found only normal fatty tissue with some encapsulation; he examined Stockman's material, and suggested that many of these sections showed little departure from the normal. The results of Abel, Siebert and Earp (1939) were equally inconclusive; low-grade inflammatory changes were reported, with early degeneration of muscle fibres. Sutro (1935) found in every case of biopsy that the nodules were composed only of fibro-fatty tissue. Albee and Campos (1939) and Jordan (1942) explained their inconclusive results by suggesting that the essential lesion might be a physicochemical change in the muscle fibres; this they called a myogelosis. Ries (1937) and Copeman and Ackerman (1944) found that the tender spots in lumbar fibrositis often were occupied by small fatty tumours whose removal frequently resulted in cure. Travell, Rinzler and Hermann (1942) suggest that structural changes occur only as a late manifestation. "It is possible", they wrote, "that the fibroblastic proliferation which has been observed is secondary to a functional disturbance and occurs only if the latter persists for a period of time."

To the present writer it seems doubtful if even the most comprehensive pathological study would prove conclusive; it seems almost certain that, in a proportion of cases, structural changes would not be observed in the tender tissues. When changes do occur in rheumatic disease, moreover, they are closely related to the changes inherent in normal deterioration and repair; and often they can be differentiated from the normal only with great difficulty. The pain occurs before the structural change, and apparently the latter is not an essential feature of the syndrome.

### Which Pains are Rheumatic?

The simplest definition of fibrositis is: "a disorder characterized by pain in the non-articular somatic structures whose cause is unknown". If the cause of such a pain is unknown, it must be presumed to be "rheumatic" in origin. If of proven rheumatic origin, the real cause still is unknown. Against this definition it might be argued that fibrositis often follows a known cause, such as injury or infection. In these instances, however, we know only the precipitating factor which has set off a trigger mechanism; but the remaining links in the chain of causation are unknown.

Many writers prefer to differentiate fibrositis from other bodily aches and pains, supposedly non-rheumatic, of unknown origin or due to psychosomatic disorder. But such a distinction is difficult to substantiate; for, seeing that the nature of rheumatism is in dispute and its cause unknown, there is no basis upon which we can classify one vague pain as rheumatic and another as "non-rheumatic, though of unknown causation". In generalized rheumatic disease, fleeting somatic pains may occur at a distance from the main lesions. The pain and tenderness may persist for only a few hours; but there is no good reason for suggesting that the rheumatic process which is the basis of these lesions differs fundamentally from the rheumatic process in the more persistent lesions.

In like manner, transient pains of unknown origin may occur in patients who are not known to be rheumatic. The symptoms signify a change in the tissues and may persist only for a few hours. The peripheral mechanism by which such pains are produced is not likely to differ from the mechanism by which fleeting pains, of proven rheumatic origin, are generated. There is therefore no scientific basis for such a fundamental distinction; and it would be pardonable to regard the fleeting pains as due to rheumatic lesions which did not become firmly established.

A familiar example is seen in the "growing pains" of children, which are thought to be rheumatic only in a minority of cases (Shapiro, 1936; Hughes, 1940). But, in our ignorance of the pathology of all these painful processes, it seems rash to assert without proof that there is a fundamental basis for differentiation between one group, rheumatism, of which we know very little, and a second group, superficially similar to the first, of which we know nothing.

### The Symptomatology of Fibrositis.

The name "fibrositis" can be applied to the majority of cases of non-articular somatic pain, either obviously rheumatic in origin or whose origin cannot be ascertained. In some of the latter a non-rheumatic cause eventually manifests itself; but in the great majority the final diagnosis (for want of a better) will be fibrositis. The chief symptom of which the patient complains is pain, usually combined with stiffness and soreness. On examination there usually will be elicited the corresponding signs, limitation of mobility and tenderness to pressure. In some cases swelling or induration is present.

*Pain.*—The pain of fibrositis may vary from a mild ache to a severe pain, with spasm of the neighbouring muscles. As a rule the pain is worse on movement, the degree of intensification depending on the structures affected. In some chronic cases, however, the pain is felt on movement only slightly (if at all); and the patient complains chiefly of a nagging ache when at rest, with an unpleasant stiffness which necessitates frequent changes of position. The pain is felt deeply and diffusely, often being difficult to locate and seeming to radiate over an area variable in extent. It radiates in patterns which tend to be constant for each region. In the limbs the pain is prone to radiate to the joints, and sometimes it seems to shoot along the whole length of the limb.

*Tenderness.*—Tenderness of the deeper structures may occur in the painful areas. It is more intense and more widespread when the pain is severe; when the pain is not severe there may be only one or two localized spots of tenderness which are not easy to identify. Usually there are two elements in the tenderness: (*a*) the localized tenderness of the myalgic lesion, (*b*) the more diffuse secondary tenderness, which disappears when the lesion is anæsthetized. The characteristics of the lesions in the special regions have been described in earlier papers (Kelly, 1942, 1944). In some cases of obscure pains the lesions can be found only after an assiduous and patient search.

*Limitation of Mobility.*—Painful muscles resent being stretched or contracted; this applies not only to the muscle which is the site of the main lesion, but also to muscles which are subject to secondary or referred tenderness. In some chronic cases there is little limitation of movement, but rather an enhanced fatigability of the muscle. Aching pain, fatigue and cramps may occur after moderate exercise. Painful cramps may be felt after holding for a few minutes a position of mild strain, and the ache and the fatigue may become unbearable. It seems not to be generally appreciated that there may be little or no restriction of movement in fibrositis. In some examples of lumbar fibrositis, and in periarthritis of the shoulder, the pain is worst when the patient is in bed.

*Swelling and Induration.*—As a rule no gross change can be discovered. Sometimes, where the subcutaneous tissues are lax, a puffy appearance can be observed, and where they are firmer induration may occur. It has been mentioned that deep indurated areas and "nodules" are not uncommon in normal people, and one should be wary of attributing significance to them. Swelling of the painful area occurs most commonly in fibrositis of the sole, which feels much thickened on walking. The anterior aspect of the knees sometimes presents a puffy appearance; and larger swellings and indurations may occur on the inner aspect of the knee above the joint-line. Diffuse induration may occur in the nuchal region; hence the name "indurative headache". In rare cases of fibrositis the painful areas present ecchymoses in addition to swelling.

*Variations in Symptoms.*—Fibrositis presents every imaginable gradation between mild and very severe pain, and between brevity of duration and intractable chronicity. Generally, attacks more sudden in onset clear up more rapidly. Acute and transient attacks are more common than the chronic forms. The lesions may be single or

multiple; they may accompany obvious rheumatic disease, or there may be no other signs. The symptoms of established fibrositis tend to wax and wane, with what seems to be a general tendency to cure. Fatigue and cold make the symptoms worse. All fibrositic lesions show a tendency to recur in the same situation.

*Local Analgesia.*—Local analgesia is useful for both diagnosis and treatment. A correctly placed injection of procaine will abolish the widely radiating pain and tenderness and the muscular stiffness. A surprising result is that the cure is lasting in about a third of the cases.

*The Structures Affected.*—The signs and symptoms vary according to the structures affected. The following classification of fibrositis was approved by the British Medical Association Committee on Chronic Rheumatic Disorders (1933): (*a*) Intramuscular and fascial. (*b*) Periarticular. (*c*) Bursal and tenosynovial. (*d*) Subcutaneous (panniculitis). (*e*) Perineuritic. When fibrositis affects the structures thus indicated, characteristic signs are added to the general picture already described. The names associated with these divisions (bursitis, tenosynovitis *et cetera*) are well established; but the creation of more "entities" by giving names to fibrositis of other structures (tendinitis, epicondylitis *et cetera*) is inadvisable.

### Ætiological Factors.

Some writers divide fibrositis into primary and secondary types. To agree with this is only to admit that some cases commence without apparent cause, while others are related in some way to preceding events. The present writer has observed fibrosis to follow a number of precipitating factors, such as injuries, local or general infections, fatigue and exposure to cold. Painful somatic lesions, in fact, may accompany or follow almost any kind of damage to body cells. Fibrositis is the result of certain precipitating factors (often unknown) acting on suitable tissues in a suitable subject. The fact that the subject is suitable often signifies that his tissues have been influenced previously by other causes. Thus we know that fibrositis is more common with advancing years, and in men who do heavy work, especially those who work out of doors (Hench *et alii*, 1935; Llewellyn, 1927); and it is commoner in the shoulders of those who labour with their arms; or in the backs of those who are required to lift heavy weights; or in the feet and knees of those whose occupation entails much walking. Previous injury or localized disease may be instrumental in predisposing a part of the body to fibrositis in later life. In about half of the cases no precipitating factor can be recognized, and about half do not present any obvious predisposing features.

The best-known predisposing factors in fibrositis have relation to prolonged overuse of a part. While Copeman (1937) held senescent fibrositis to be especially intractable because of the degenerative changes and the fibrosis, others regard age as "but the sustained opportunity for mild long-continued trauma" (Hench *et alii*, 1936). Prolonged overuse of a part, without doubt, hastens the normal processes of degeneration. But the question is not so clear-cut when it relates to moderate every-day use, either as a precipitating or as a predisposing factor. A severe pain, which later becomes intractable, may commence during an ordinary movement. Lumbago, said Poynton and Schlesinger, "is a perplexing disorder, especially when it suddenly attacks a healthy man when bending down to lace up his boots. The usual explanation is chill and sudden strain in a person with a gouty or rheumatic constitution, but are the factors involved quite as simple as this?"

In such a case, there can be little doubt that the lesion resulted from the simple movement; but we do not know what other factors, favourable to its development, were present. A localized area of muscle suddenly has become painful; and, in the absence of evidence of constitutional and predisposing factors, we must attribute it to purely local and fortuitous causes. We may, for instance, assume that a few muscle fibres were caught "off their guard", and through acting at a mechanical disadvantage became damaged. When the case has dragged on to become chronic lumbago, there still is no necessity to postulate unknown constitutional factors; for we know that local conditions tend to prolong the duration of a muscular lesion. Muscle, whose excitability is enhanced by the painful lesion, finds it more difficult to secure the rest which is necessary for the healing of the lesion; and thus "myalgic" lesions tend to perpetuate themselves. The persistence of the lesion would be rendered more likely by previous degeneration of the tissues, owing to age or attrition.

Copeman (1943) made the interesting observation that, following such infectious diseases as influenza, tender spots appear in the muscles, which later may function as "myalgic" lesions in an attack of fibrositis. Perhaps there is some relation here to the bodily pains and aches which accompany many febrile illnesses. If such a patient is carefully examined, it can often be proved that the acute pains are due to localized lesions in muscle, which produce as secondary effects radiating pain and deep tenderness. In a number of such cases the writer has been able to abolish the radiating pain by means of a small injection of procaine, correctly placed. The acute lesion in febrile myalgia thus behaves exactly like a fibrositic lesion. When it persists and becomes chronic, by general agreement it is called fibrositis (post-influenzal, post-scarlatinal *et cetera*).

It is not a coincidence, therefore, that latent lesions appear in somatic tissues after febrile illnesses (as described by Copeman). It is but reasonable to argue that the acute febrile myalgic lesions have not cleared up entirely, but have persisted as foci of latent activity.

### Psychosomatic Rheumatism.

Many observers doubt the reality of pains which are not accompanied by gross physical signs, and most writers on rheumatism claim that they can differentiate a psychosomatic group of painful disorders, which cannot rightly be called rheumatic. There is every form of gradation in this rejection of an organic basis for the pain. Halliday (1942) holds that the pain, stiffness and soreness are merely symbolical, signifying the patient's lack of harmony with his environment. Most other writers allow the diagnosis of fibrositis if tender spots can be found, but in the absence of this sign they regard the condition as psychogenic (Hutchison, 1942; Heaton, 1944; Flind and Barber, 1945). Suspicion is enhanced: (i) if the pain shows a tendency to wander; or (ii) if it does not correspond with the distribution of a peripheral nerve. Essential to the diagnosis is a history of psychic strain.

But the difficulties are many which inevitably beset the clinician who tries to sustain such a clear-cut differentiation. The failure to demonstrate tenderness in the painful area is not of great significance, for pain from a small muscular lesion (artificial or fibrositic) may radiate to distant parts; and these parts may not be tender to pressure (Kellgren, 1938, 1939; Lewis, 1942; Inman and Saunders, 1944). The existence of tenderness, therefore, should not be denied until some time has been devoted to a careful examination of the soft tissues by palpation. But those who uphold the psychosomatic hypothesis hold such a painstaking survey to be inadvisable, because of the probability of "fixing" the patient's symptoms by suggestion. For the same reason, some authorities hold the eliciting of a careful history to be injudicious. The late Sir Arthur Hurst (1943) spoke of "iatrogenic" disorders—diseases brought on or worsened by the error of the practitioner in attributing too much importance to the patient's symptoms.

Though we are told that "wandering" is characteristic of psychogenic pain, it occurs also in deep somatic pain. Lewis and Kellgren have demonstrated that the pain from artificial deep lesions radiates over variable areas, that it fluctuates in intensity, and that it never corresponds with the distribution of cutaneous nerves.

After observation of a number of patients whose pains had been diagnosed as functional, the writer finds increasing difficulty in laying down reliable standards for differentiation. Pain due to organic disease has been diagnosed as psychogenic before the underlying cause manifested itself. Recent examples are: a carcinoma of the pancreas causing epigastric discomfort; a secondary growth from renal carcinoma, causing pain in the thigh; and spinal metastases from carcinoma of the suprarenal causing low back pain. In the third case the patient became insane before he died.

XUM

This was thought to confirm the diagnosis of functional pain, but the autopsy revealed cerebral metastases also.

The diagnosis of psychosomatic pain does not rest upon positive evidence; rather it depends on lack of evidence. The greater the psychiatric bias of the physician, the larger will be the proportion of his cases which acquire the label. From the majority of normal people can be elicited a history of worries, strains or conflicts, which easily may be magnified to assume the proportions of "psychic trauma"; and so it is rare that the clinician who "spot-diagnoses" psychogenic pain fails to elicit a confirmatory history. Writing of juvenile rheumatism, D. Hubble (1943) stated: "To write and talk of 'psychosomatic' disorders is to assume a bridge whose existence we but dimly discern and of whose nature we are ignorant."

There is little doubt that rheumatic disease may be brought on by severe nervous exhaustion or prolonged anxiety. The onset of rheumatoid arthritis is preceded, too frequently for mere coincidence, by just such a history. This is an example of somatic disease caused by a mechanism whose nature is unknown, for which mental exhaustion may be as potent a precipitating factor as is physical exhaustion. In such "psychosomatic" diseases, the precipitating factor is psychic, but the effects are somatic, and the link between psyche and soma is unknown. Sir Clifferd Allbutt (1910) described somatic pain as a symptom of neurasthenia, but he did not suggest that the pains were in any sense unreal. After prolonged anxiety, one patient may suffer from asthma, another from peptic ulcer, another from *alopecia areata* (Hubble and Rogerson, 1944), and another from rheumatoid arthritis. In all of these examples the visible abnormality is in the periphery and not in the central nervous system. And when somatic pain accompanies neurasthenia, it seems reasonable to suggest that there has been induced an abnormality of the somatic tissues. "All pains are peripherally induced and subjectively appreciated", wrote Meyer Solomon (1917) when, after an erudite discussion, he gave a negative answer to the question: Are there subjective pains? "If the patient complains of a pain", he said, "he really has a pain."

Psychosomatic pains, it is suggested, cannot always be differentiated from somatic pains, for the pain itself always is somatic and the same mechanism is used. It is an error to regard the pain in any sense as unreal or symbolical; such an inviting hypothesis brings to a diagnostically destitute physician the double comfort of laying upon the patient both the blame for being ill and the responsibility of curing himself. "We should not hide our ignorance", wrote the late Sir E. Farquhar Buzzard (1929), "behind such diagnoses as psychalgia or functional pain." In addition to leading sometimes to serious errors in diagnosis, such a diagnosis contravenes the basic medical maxim that a patient's disorder must be considered organic unless proved otherwise. It is suggested that, if the use of the word "fibrositis" is allowable, it is not less applicable to many of the so-called "psychosomatic" pains than to other pains of doubtful causation.

## Discussion.

The object of this paper is to demonstrate upon what insecure grounds we undertake to differentiate and classify the common pains of daily practice. There is no useful classification except to call them all by the same name, and for this "fibrositis" is as useful a term as any other. The feature common to them all is pain, which tends to occur in frequently repeated patterns. Pain is the patient's chief complaint, and often the only one. Even when the disorder is obviously rheumatic in origin, pain often is the only sign or symptom. To quote René Leriche (1939), pain "constitutes the whole disease, which, failing it, would not exist at all". It must follow therefore that this group of disorders (including rheumatism of every variety) must remain a complete mystery until light has been thrown upon the functional pathology of spontaneous somatic pain. Until we know what pain is, there is no basis upon which a classification of these pains can even be attempted; and it is the purest surmise to say that fibrositis (rheumatic pain) differs from "psychosomatic pain", and that both differ from other pains whose cause is unknown.

A great deal of evidence has accumulated in recent years which suggests that the deep pain apparatus is closely linked to the fibrous and supporting structures (Weddell and Harpman, 1940; Lewis, 1942). By the use of selective local analgesia, it can be shown that it is not the substance of the muscle, tendon and bone which becomes tender to pressure in rheumatic and other diseases, but the layer of fibrous tissue which invests these structures (Kelly, 1945, 1946).

When tenderness is the sole objective sign, the structure involved is fibrous tissue. In the next stage of the disorder, when structural changes characteristic of rheumatism can be recognized, they, too, occur at first in the fibrous and supporting structures. The pain and tenderness, therefore, must signify a functional change in the cells of fibrous tissue, which, when prolonged and intensified, leads to the fibroblastic reaction which is characteristic of rheumatism.

Clinically, somatic pain and fibrositis present a number of gradations occupying a sort of spectrum:

(*a*) At the red end of the spectrum is pain without any physical signs. Possibly undiscovered "myalgic lesions" are responsible.

(*b*) Localized myalgic lesions can be identified upon careful examination. These are responsible for the radiating pain; there may or may not be limitation of movement.

(*c*) The discrete lesions may give rise to wider areas of diffuse secondary or "referred" deep tenderness, usually with limitation of movement. The signs disappear when the offending lesion is anæsthetized.

(*d*) Swelling or induration may occur in the diffusely tender areas, owing to reflex outpouring of fluid into the tissues.

(*e*) If certain somatic tissues are involved, this œdematous reaction takes on special characters, for example, roughening of membranes or effusion into synovial cavities (bursitis, tenosynovitis, dry pleurisy, hydrarthrosis *et cetera*).

(*f*) If the fibrous tissues involved are closely associated with specialized organs, the latter may be damaged secondarily by the products of rheumatic inflammation (carditis, interstitial neuritis, muscular atrophy *et cetera*).

(*g*) If the process is subacute or chronic, the effects of repair and fibrosis may proceed coincidently with the active rheumatic process, and secondary modifications of structure ensue. This is the violet end of the spectrum.

Like the various tints of the spectrum, these denominations merge into each other imperceptibly, and the change from the red to the violet end is steady and gradual. It is difficult indeed to say at what stage the process becomes "fibrositis", and at what other stage "arthritis". In a patient with multiple rheumatic lesions, there may be one or more lesions to represent each "tint", including stage (*a*)—pain unaccompanied by physical signs to account for it. Or a patient who has suffered from acute rheumatism in a joint may complain of aching pain in the joint long after all swelling, tenderness and limitation of movement have disappeared—symptoms which Flind and Barber (1945) would regard as an hysterical prolongation of a former rheumatic condition. It is likely that the mechanism of the underlying disorder is the same in all cases. The safest course is to regard the pain in all of them as "peripherally induced and subjectively appreciated", and to label them all fibrositis.

## Summary.

1. Clinically, fibrositis is characterized by pain, usually accompanied by tenderness and muscular stiffness; but examination of the affected tissues has not revealed a constant pathological picture. It is suggested, moreover, that the results of such procedures always will be inconclusive.

2. Fibrositis is not a disease, but a special kind of reaction in fibrous tissue, which may result from a number of different precipitating factors. In its early stages, this reaction manifests itself by disorders of tissue function rather than by recognizable structural changes.

3. When structural changes occur, they are those of acute or chronic rheumatism. It is suggested that fibrositis is the basic lesion, common to all rheumatic diseases, and

producing special signs and symptoms only when it invades special tissues.

4. There is no reliable basis upon which fibrositis of the mildest kind can be differentiated from other pains of unknown or of psychosomatic origin. All pains should be regarded as peripherally induced until proved otherwise.

5. The term "fibrositis" should be retained to cover all these common pains of daily practice, which display constant or recognizable patterns; and which present every gradation of severity, from vague pains to advanced rheumatic disease.

### Bibliography.


O. Abel, W. J. Siebert and R. Earp: "Fibrositis", *Journal of the Missouri Medical Association*, Volume XXXVI, 1939, page 435.

F. H. Albee and O. P. Campos: "Low Back Pain and Allied Conditions", *The American Journal of Surgery*, Volume XLIII, 1939, page 386.

T. C. Allbutt: "Neurasthenia", T. C. Allbutt and H. D. Rolleston's "System of Medicine", Volume VIII, 1910, page 727.

W. Boyd: "Pathology of Internal Disease", 1940.

W. Bruce: "Lumbago", *British Medical Journal*, Volume I, 1904, page 217.

E. Farquhar Buzzard: "Discussion on Abdominal Pain", *The Lancet*, Volume II, 1929, page 226.

M. Cassidy: "The Etiology and Treatment of Osteo-Arthritis and Rheumatoid Arthritis", *Proceedings of the Royal Society of Medicine*, Volume XVII, 1923, page 4.

D. H. Collins: "The Pathological Anatomy of Rheumatic Fever", *British Journal of Rheumatism*, Volume I, 1938, page 88; "Fibrositis and Infection", *Annals of the Rheumatic Diseases*, Volume II, 1940, page 114.

W. S. C. Copeman: "On Fibrositis—the English View", *Acta Rheumatologica*, Volume IX, 1937, page 6 (cited by Hench *et alii*, 1939); "Clinical Contribution to Study of Etiology of Fibrositic Nodule", *Annals of the Rheumatic Diseases*, Volume III, 1943, page 222; "Observations on the Natural History of Rheumatic Fever", *ibidem*, Volume IV, 1944, page 11.

W. S. C. Copeman and W. L. Ackerman: "'Fibrositis' of the Back", *The Quarterly Journal of Medicine*, Volume XIII, 1944, page 37.

W. S. C. Copeman and L. G. C. Pugh: "Effects of Artificial Dehydration in Rheumatism", *The Lancet*, Volume II, 1945, page 553.

Editorial (Annotation), "Fibrositis, Organic and Inorganic", *The Lancet*, Volume II, 1942, page 652.

J. Flind and H. S. Barber: "The Psychogenic Basis of Some So-called Rheumatic Pains", *The Quarterly Journal of Medicine*, Volume XIV, 1945, page 57.

R. G. Gordon: "Osteo-Arthritis and its Concomitants", *British Medical Journal*, Volume II, 1935, page 1083.

W. Gowers: "Lumbago, its Lessons and Analogues", *British Medical Journal*, Volume I, 1904, page 117.

J. L. Halliday: "The Obsession of Fibrositis", *British Medical Journal*, Volume I, 1942, page 164.

T. G. Heaton: "Arthralgia", *Canadian Medical Association Journal*, Volume L, 1944, page 515.

P. S. Hench, W. Bauer, A. A. Fletcher, D. Ghrist, F. Hall and P. White: First Rheumatism Review: "The Present Status of the Problem of 'Rheumatism': A Review of Recent American and English Literature on 'Rheumatism' and Arthritis", *Annals of Internal Medicine*, Volume VIII, 1935, page 1315; Second Rheumatism Review: "The Present Status of the Problem of 'Rheumatism' and Arthritis: Review of American and English Literature for 1934", *ibidem*, Volume IX, 1936, page 883.

P. S. Hench, W. Bauer, M. H. Dawson, F. Hall, W. P. Holbrook and J. A. Key: Fifth Rheumatism Review: "The Problem of Rheumatism and Arthritis: Review of American and English Literature for 1937", *Annals of Internal Medicine*, Volume XII, 1939, pages 1005 and 1295.

P. S. Hench, W. Bauer, M. H. Dawson, F. Hall, W. P. Holbrook, J. A. Key and C. McEwen: Seventh Rheumatism Review: "The Problem of Rheumatism and Arthritis: Review of American and English Literature for 1939", *Annals of Internal Medicine*, Volume XIV, 1941, pages 1383 and 1631.

P. S. Hench, W. Bauer, E. Boland, M. H. Dawson, R. H. Freyberg, W. P. Holbrook, J. A. Key, L. M. Lockie and C. McEwen: Eighth Rheumatism Review: "Rheumatism and Arthritis: Review of American and English Literature for 1940", *Annals of Internal Medicine*, Volume XV, 1941, page 1002.

D. Hubble: "The Nature of the Rheumatic Child", *British Medical Journal*, Volume I, 1943, pages 121-154.

D. Hubble and C. H. Rogerson: "Anxiety, Alopecia Areata, Neurofibromatosis, Auricular Fibrillation", *British Medical Journal*, Volume I, 1944, page 486.

L. Hughes: "Acute Rheumatism", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1940, page 423.

A. Hurst: "Foreign Letters", *The Journal of the American Medical Association*, Volume CXXI, 1943, page 879 (abstract).

J. H. Hutchison: "Non-Articular Rheumatism in the Army: Symptomatology, Etiology, Treatment", *Glasgow Medical Journal*, Volume XIX, 1942, page 33.

V. T. Inman and J. B. de C. M. Saunders: "Referred Pain from Skeletal Structures", *Journal of Nervous and Mental Disease*, Volume XCIX, 1944, page 660.

H. H. Jordan: "Myogeloses: Significance of Pathological Conditions of Musculature in Disorders of Posture and Locomotion", *Archives of Physical Therapy*, Volume XXIII, 1942, page 36.

J. H. Kellgren: "Observations on Referred Pain Arising from Muscle", *Clinical Science*, Volume III, 1938, page 175; "Pain Arising from Deep Somatic Structures", *ibidem*, Volume IV, 1939, page 35.

M. Kelly: "Lumbago and Abdominal Pain", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1942, page 311; "New Light on the Painful Shoulder", *ibidem*, Volume I, 1942, page 488; "Headaches, Traumatic and Rheumatic: The Cervical Somatic Lesion", *ibidem*, Volume II, 1942, page 479; "Pain in the Chest: Observations on the Use of Local Anæsthesia in its Investigation and Treatment", *ibidem*, Volume I, 1944, page 4; "Peri-articular Fibrositis of the Knee and the Value of Local Analgesia", *ibidem*, Volume I, 1944, page 286; "Pain in the Forearm and Hand due to Muscular Lesion", *ibidem*, Volume II, 1944, page 185; "The Nature of Fibrositis. I. The Myalgic Lesion and its Secondary Effects", *Annals of the Rheumatic Diseases*, Volume V, 1945, page 1; "The Nature of Fibrositis. II. A study of the Causation of the Myalgic Lesion (Rheumatic, Traumatic, Infective)", *ibidem*, 1946 (in the press).

R. L. J. Llewellyn: "Aspects of Rheumatism and Gout: Their Pathogeny, Prevention and Control", 1927.

T. Lewis: "Pain", 1942.

R. Leriche: "The Surgery of Pain", 1939.

F. J. Poynton and B. Schlesinger: "Recent Advances in the Treatment of Rheumatism", 1937.

L. G. C. Pugh and T. A. Christie: "A Study of Rheumatism in a Group of Soldiers with Reference to the Incidence of Trigger Points and Fibrositic Nodules", *Annals of Rheumatic Diseases*, Volume V, 1945, page 8.

E. Ries: "Epi-Sacro-Iliac Lipoma", *American Journal of Obstetrics and Gynecology*, Volume XXXIV, 1937, page 490.

Editorial, "Sciatic Pain", *British Medical Journal*, Volume II, 1941, page 698.

M. J. Shapiro: "The Natural History of Childhood Rheumatism in Minnesota", *Journal of Laboratory and Clinical Medicine*, Volume XXI, 1936, page 564.

M. Solomon: "Are There Subjective Pains?" *Medical Record*, Volume XCII, 1917, page 314.

R. Stockman: "The Causes, Pathology and Treatment of Chronic Rheumatism", *Edinburgh Medical Journal*, Volume XV, 1904, pages 107 and 223; "Rheumatoid Arthritis", in "Nelson's Loose-Leaf Medicine", 1920; "Chronic Muscular Rheumatism and Panniculitis", *British Medical Journal*, Volume I, 1928, page 293.

C. J. Sutro: "Subcutaneous Fatty Nodules in Sacro-Iliac Area", *The American Journal of the Medical Sciences*, Volume CXC, 1935, page 883.

H. F. Swift: "The Nature of Rheumatic Fever", *Journal of Laboratory and Clinical Medicine*, Volume XXI, 1936, page 551.

"The Causation and Treatment of Arthritis and Allied Conditions", report of committee appointed by the British Medical Association, *British Medical Journal*, Volume I, 1933, page 1033.

C. H. Traeger: "Fibrositis", *Medical Clinics of North America*, Volume XXI, 1937, page 1797.

J. Travell, S. Rinzler and M. Hermann: "Pain and Disability of the Shoulder and Arm: Treatment by Intramuscular Infiltration with Procaine Hydrochloride", *The Journal of the American Medical Association*, Volume CXX, 1942, page 417.

F. M. R. Walshe: "Some Principles of Reform in Medical Education", *British Medical Journal*, Volume I, 1944, page 173.

G. Weddell and J. A. Harpman: "The Neurohistological Basis for the Sensation of Pain Provoked from Deep Fascia, Tendon and Periosteum", *Journal of Neurology and Psychiatry*, Volume III, 1940, page 319.


## LUMBAGO.

By JAMES H. YOUNG, M.D. (Adelaide), M.R.A.C.P.,
*Perth*.

### The Fibrositic Theory.

FORTY years ago Sir William Gowers[1] delivered a lecture with the title "Lumbago: Its Lessons and Analogues". He expressed the opinion that lumbago was an inflammatory affection of the fibrous tissue of the lumbar muscles—that is, "fibrositis". As far as I am aware, he introduced this term into medicine. His views gained general acceptance throughout the British Empire, but fibrositis has never been a popular diagnosis in America.

Gowers stated that the pain of lumbago was felt only when the lumbar muscles were contracted or extended, and he therefore inferred that the pain was muscular in origin. At about this time the muscle spindles had recently been described. They were the only known sensory end organs in muscle, and it was therefore thought that they transmitted the painful impulses from the muscles. As the muscle spindles were associated with the fibrous tissue of muscle, Gowers concluded that lumbago was a disease of the fibrous tissue of muscle. He explained the relationship between lumbago and sciatica on the basis of a spread of

the pathological process from the fibrous tissue of the back along fascial planes to the sheath of the sciatic nerve and sometimes into the nerve itself; in the latter case there was evidence of impairment of conduction in the nerve fibres. He considered that sciatica was an inflammatory condition (though he did not give any reasons for his belief), and argued that the lumbago which preceded the sciatica must also have been inflammatory. He further stated that the whole process ran the acute course of an inflammatory condition and that it spread along fascial planes as did other inflammatory conditions.

Such was the reasoning which led Gowers to believe that lumbago was an inflammatory lesion of fibrous tissue. He insisted on the inflammatory nature of the process, yet he noted that it differed from ordinary inflammation in that there was no indication of the formation of inflammatory products and the onset might be extremely sudden. He considered that the extreme suddenness of the onset of lumbago was more often apparent than real:

> There has before been some slight definite discomfort in the muscles, and then some peculiar action causes an extreme stimulation of the muscles which had been gradually becoming over-sensitive. Hence the morbid process may be much less sudden than is its manifestation; the production of the pain is sudden, but the condition on which it depends has been gradually developed.

He went on to draw an analogy with stiff neck and brachial neuritis, and believed that these were also fibrositic; but he concluded his paper with the following words:

> At present we are without any direct evidence of the real nature of these affections, such as can only be furnished by the microscope and modern methods of discerning that which it reveals.

These words make me feel sure that Gowers would have had very different opinions if he had known of the existence of pathological changes in the intervertebral disk and of the radiological changes that occur in disk disease.

### The Diskogenic Theory.

For some years it has been known that certain patients with low-back pain eventually developed sciatica due to protrusion of an intervertebral disk, and it has been obvious that in these patients the low-back pain must also have been diskogenic. In 1944 Dandy[2] described disk lesions found at operation in patients suffering from recurring attacks of low-back pain. Key,[3] as a result of surgical experience, believed that an intervertebral disk lesion was the cause of over 90% of idiopathic low-back pain. Clark found disk lesions at operation in patients of mine suffering from low-back pain, and I[4] expressed the opinion that bending and lifting injuries usually damaged the disk, that degeneration of an intervertebral disk was the most common cause of chronic low-back pain, and that acute lumbago was due to a disk lesion. Burns and Young[5] stated that the chief cause of recurrent low-back pain was protrusion of an intervertebral disk, and Cyriax[6] thought that lumbago was due to bruising of the spinal *dura mater* from a momentary protrusion. There is a wide divergence of opinion between these views and those originally put forward by Gowers.

It is not sufficient to state that fibrositis does not exist, purely and simply because microscopic evidence of its existence is lacking. If the diskogenic theory of the origin of lumbago is to be accepted, it is necessary to prove that the fibrositic theory is based upon false reasoning and to bring forward sound argument in favour of the diskogenic view. This involves a consideration of Gowers's paper to show where his reasoning was false, and a careful analysis of the signs of lumbago to prove that the anatomical structure affected is in fact the intervertebral disk. It is also necessary to suggest pathological lesions which may produce lumbago, the existence of such lesions to be based upon knowledge and not upon theory. These are the objects of this paper.

### The Anatomical Site of the Lesion in Lumbago.

In most medical conditions it is possible to determine the anatomical site of the lesion by a careful consideration of the signs, and to determine the pathological type of lesion by a careful consideration of the symptoms. Let us then apply this type of reasoning to the problem of lumbago.

Take a patient with lumbago and inspect him in the standing position. If there is a lateral flexion deformity (that is, sciatic scoliosis—a term which is better avoided, for this deformity may occur in the absence of sciatica), then the patient almost certainly has a protruded intervertebral disk.[4][5] Such patients, in whom the pathological condition has often been proved at operation, should not be classed as suffering from acute lumbago, and only those in whom no deformity is visible are considered in this paper.

Ask the patient to bend backwards. He will usually complain of pain, and the more he extends, the more pain he will suffer. Now this pain cannot entirely be due to contraction of the sacrospinales. Wood Jones[7] has pointed out that certain movements of joints are not carried out by muscles acting as prime movers, but are due to the action of gravity, which acts as the prime mover, *plus* gradual relaxation of the antagonists. This is the "action of paradox". Extension of the spine is initiated, but only initiated, by contraction of the sacrospinales. At this stage the *recti abdominis* are relaxing. As soon as the first few degrees of the movement have been executed further extension is carried out by gravity; the sacrospinales then cease to act, while the antagonists (the *recti abdominis* and the psoas muscles) contract to prevent the trunk from toppling backwards. Thus the pain which is experienced at the beginning of extension may be due to muscular contraction, but not the pain which is felt on further extension of the spine.

Next, make the patient lie face down on an examination couch and lift his lower limbs off the couch, thus extending his spine passively. He will complain of acute pain, and yet the sacrospinales have not even initiated the movement. Thus the pain which is felt on extension of the spine is not due to contraction of the muscles.

Flexion of the spine is painful and limited, the lumbar part of the spine remaining fixed in lordosis. The sacrospinales and glutei contract during flexion (the action of paradox) to prevent the trunk from toppling forwards. The pain which is experienced on flexion may be due partly to stretching of the sacrospinales or the glutei or to contraction of these muscles, as can be shown by the improvement which sometimes follows the injection of a local anæsthetic agent. More often such injection makes no difference, and then the pain which is experienced on flexion cannot be muscular in origin. Thus it appears that there are two main causes of pain on flexion in patients suffering from lumbago: (i) pain which is not relieved by infiltration of the muscles with a local anæsthetic agent and which is not muscular in origin, and (ii) pain which is relieved (seldom abolished) by such infiltration. In the latter case the pain would appear to be generated at least in part in the muscles; but diminution of the pain following infiltration of the muscles with procaine does not necessarily mean that the primary cause of the pain is in the muscles, for such improvement may occur in the presence of proved disk lesions. In such cases the improvement can be explained by the work of Elliott,[8] who has shown that a disk lesion, by involving motor fibres in nerve roots, either directly or reflexly, may lead to areas of spasm in muscles. These areas may be painful, and the injection of a local anæsthetic agent may relieve the pain.

Thus we see that the pain which is experienced on flexion of the spine is not usually due to stretching or contraction of the muscles, and even when it appears to be generated in part in the muscles, the primary cause of the pain may lie outside the muscles.

Gowers's statement that the pain is felt only on contraction or extension of the muscles cannot therefore be substantiated by a careful consideration of the action of the lumbar muscles or of the physical signs in a patient suffering from lumbago, and his assumption that the pain is muscular in origin is therefore based upon false reasoning.

If, then, the pain is not muscular in origin, in what structure does it arise? Does it arise in the subcutaneous fat, in the fascial sheaths of the muscles, in ligaments, in bones, in the spinal *dura mater*, in the interlaminar joints or in the intervertebral disks?

One can hardly imagine that the subcutaneous fat is subjected to much stretching during flexion or to much compression during extension of the spine, and anyone who has noticed œdema of the hand in a patient with brachial neuritis would not be surprised to find congestion and œdema of the fat globules in that condition or in the fat globules of the back in patients suffering from lumbago. Congestion and œdema occasionally occur in the foot after a disk operation and would appear to be due to lack of vasoconstrictor impulses.

Evidence has already been presented to show that the pain of lumbago is not usually muscular in origin, and this evidence can also be used to argue that the pain does not arise in the muscle sheaths or for that matter in any herniations of fat through these sheaths. Furthermore, one notes that the point of maximum tenderness in lumbago is over the interspinous ligament and not over the muscles, over their sheaths or over any fat herniations through these sheaths.

Anatomically the anterior and posterior common ligaments may be considered to be parts of the intervertebral disks, and it would hardly be possible to distinguish injury or disease of these ligaments from injury or disease of the disk; it would also seem likely that pathological processes affecting one would soon affect the other. Any pain arising in these ligaments may therefore be considered to be diskogenic.

The *ligamenta subflava* and the intertransverse ligaments are not fully stretched in flexion or compressed in extension; but of course if they were inflamed, then they could be fully stretched or compressed by these movements. One notes, however, that the maximum tenderness in lumbago is usually over the interspinous ligament and not over the *ligamenta subflava* or over the intertransverse ligaments. Inflammatory changes have never been reported in the *ligamenta subflava*. Thickening of these ligaments is present when the laminæ lie more horizontally than usual, and may occur as part of a general ligamentous shortening which occurs around a degenerated disk; this is nature's method of overcoming the abnormal mobility of such a disk.

If the patient lies prone on the examination couch with a firm pillow under the abdomen, and if pressure is applied over each interspinous ligament, tenderness will usually be found either between the fourth and fifth lumbar or between the fifth lumbar and first sacral spinous processes, and less commonly at other levels. This tenderness is exactly similar to the tenderness which occurs over proved disk lesions, and may be of two types. (i) Sometimes the ligament is tender on light pressure, and the tenderness can be abolished by infiltration with a local anæsthetic agent. It can also be abolished by this procedure in patients with proved disk lesions, so that the relief of this type of tenderness by local anæsthetic injection does not necessarily mean that the cause of the tenderness lies in the ligament. Presumably this tenderness on light palpation is due to hyperæsthesia of the ligament. (ii) More often tenderness is found only on firm pressure over the affected ligament, and this tenderness and the pain which is experienced on flexion and extension of the spine are not abolished by the injection of procaine in and around the ligament. Thus the interspinous ligament is not the site of the pain in lumbago. I believe that the pain which is produced by firm pressure over the interspinous ligament is due to movement occurring between the vertebræ when such pressure is applied.

Bony lesions can hardly be presumed to be the cause of lumbago. They do not cause pain on movement, and in patients suffering from lumbago there are no radiological signs indicating pathological changes in bone. Osteophytic lipping, when present, is usually found at the margins of a thin disk.

That the spinal *dura mater* plays some part in the production of the symptoms in lumbago is sometimes obvious. The neck flexion and sciatic stretching tests often aggravate the pain of sciaica, and they do so by stretching the nerve roots through the spinal *dura mater* or the sciatic nerve. It is not well known that neck flexion and sciatic stretching sometimes cause pain in a patient suffering from lumbago; presumably they do so by stretching the spinal *dura mater* or by stretching adhesions between the spinal *dura mater* and a disk. It must therefore be conceded that the spinal *dura mater* may play some part in the production of symptoms, but I believe that it is only a secondary part, because neck flexion and sciatic stretching do not usually cause pain in a patient suffering from lumbago. (In passing, it is interesting to note that, in a patient suffering from stiff neck, the pain is sometimes aggravated by straight-leg raising.) Relief of the pain in lumbago by epidural anæsthesia does not prove that the pain arises in the dura, because the local anæsthetic agent bathes the nerve roots and thus blocks off painful impulses from every structure in the back.

By a process of exclusion, therefore, we are forced to conclude that the pain of lumbago arises in an interlaminar joint or in an intervertebral disk.

Now lumbago is a common malady, and if it was due to disease of an interlaminar joint one would expect to find radiological evidence of disease of these joints in a proportion of patients suffering from low-back pain. In point of fact, radiological evidence of disease of these joints is rare; I have seen arthritis of an interlaminar joint only twice in my last 500 X-ray films. On the other hand, radiological evidence of disk disease is common, occurring in some 39% of these X-ray films.

Patients suffering from sciatica due to a proved herniation or degeneration of a disk often have a history of attacks of lumbago. Now it is a rule of medicine not to diagnose two conditions if one lesion will explain the whole story, and all the symptoms and signs of lumbago can be explained by a disk lesion. It is therefore reasonable to assume that in such cases the previous attacks of lumbago were also diskogenic. (I have actually followed up one patient from her first attack of lumbago to the operating table, where she was proved to have a degenerated lumbar disk.) Disk lesions have now been found at operation in a number of patients suffering from recurring attacks of low-back pain without sciatica. In those attacks of lumbago which have not yet been—and we hope will not be—followed by sciatica or by a surgically proved disk lesion, all the symptoms and signs can be explained on the basis of a disk lesion, and they cannot be explained by a lesion in any other situation.

I therefore conclude that, in lumbago, the anatomical site of the lesion is in an intervertebral disk. Similar reasoning leads one to the same conclusion about stiff neck.

### The Nature of the Pathological Lesion in Lumbago.

If we accept the view that lumbago is diskogenic, we must be prepared to suggest the type of lesion which may be present. Now what pathological lesions are known to occur in the lumbar disks? These are rupture, hæmorrhage, œdema, degeneration and herniation. A careful consideration of the patient's history should enable us to determine which of these lesions is the most likely explanation of the symptoms in lumbago.

Gowers considered that lumbago was an inflammatory condition, but he had no real evidence to support his opinion. In fact, he pointed out that "the features of the malady differ from those of ordinary inflammation" and that there was no evidence of the formation of inflammatory products. Today we know that lumbago differs from every other known inflammatory condition in that no fever, no leucocytosis or no increase of the blood sedimentation rate is present. Certainly lumbago often runs an acute course like an inflammatory condition; but this is no reason for regarding it as such, and the spread of the pain from the back to the lower limb is much more easily explained on the basis of a disk lesion (which only has to extend a fraction of an inch in order to involve a lumbo-sacral nerve root) than on the basis of an inflammatory condition of the fibrous tissue. Another of Gowers's

difficulties in explaining lumbago as an inflammatory lesion was the extreme suddenness of onset which is sometimes a feature. Admittedly he stated that this was "often apparent, not real", and that there had "before been some slight definite discomfort". This previous discomfort is easily explained as being due to degeneration of a disk, evidence of which can sometimes be seen in the radiograph.

An extremely sudden onset is usually interpreted in medicine as indicating either a mechanical or a vascular lesion. The former is probably more often the cause of lumbago than is generally realized. The influence of trauma can be appreciated only if a careful history is taken. For example, a patient who may have had some previous slight discomfort may wake up feeling perfectly well. His muscles are relaxed and off guard; he twists in order to get out of bed, and as he does so, he feels a sudden acute pain in the back. Now this twisting is of course an example of indirect violence. Could not such violence injure a disk, and especially a degenerated disk? Many patients have been followed from the time of an original low-back strain due to indirect violence right through to the operating table, upon which they have been proved to have a disk lesion. Such indirect violence may cause a partial rupture of the cartilage plate or of the annulus, or as Cyriax[6] has suggested, it may produce "a momentary posterior displacement of a movable piece of intra-articular fibrocartilage", thus bruising the *dura mater*. Such bruising may explain those cases of lumbago in which the pain is aggravated by neck flexion or by sciatic stretching; but these signs may also be produced by stretching adhesions between the disk and the dura.

Can we postulate that some cases of lumbago are due to hæmorrhage into a disk? Hæmorrhage from the blood vessels of the vertebral bodies may rupture through the cartilage plates into degenerated disks. Such hæmorrhages have been observed *post mortem*, and Beadle[9] states that they are usually traumatic, but some of them may be spontaneous. Dandy,[10] in describing the "concealed disk", stated that the interior of such a disk was sometimes soft and brown. Beadle had previously described a "brown degeneration", and it is tempting to suggest that this brown discoloration has resulted from previous hæmorrhage. It is therefore possible that an occasional case of lumbago may be due to spontaneous hæmorrhage into a disk.

A rapid onset as opposed to an extremely sudden onset may result from œdema of a piece of a disk. Œdema has been observed, and Deucher and Love[11] have suggested that such œdema may come and go, the appearance and disappearance of the œdema corresponding to the exacerbation and remission of symptoms of herniation of a disk. Œdema of a piece of disk would be more likely to produce symptoms of herniation of a disk than of lumbago. The relationship of the onset of pain to changes of weather, draughts and getting wet seems more than fortuitous, and one wonders if variations of temperature and of atmospheric pressure can produce œdema of a disk.

Degeneration of a disk is, as far as we know, a chronic condition, and one would not expect acute symptoms to result from the degeneration *per se*. I think that degeneration of a disk accounts for the previous discomfort so often experienced by patients who have lumbago, but not for the acute attack itself, which is more likely to be due to superadded trauma, hæmorrhage, displacement or œdema.

### The Treatment of Lumbago.

If we accept the view that lumbago is a disk lesion, and I believe that all the available evidence compels us to do so, then we must treat it as a disk lesion by adequate rest and support and by the avoidance of anything which may increase the damage. Details of this treatment have been described previously.[12]

### Conclusion.

The reasons which Gowers advanced for the existence of fibrositis cannot be substantiated by a careful consideration of the action of the lumbar muscles or of the symptoms and signs in a patient suffering from lumbago.

The existence of fibrositis as an entity is doubtful, and the clinical features of lumbago cannot be explained by such a lesion.

Is not lumbago better explained by a lesion of an intervertebral disk?

### Acknowledgement.

I am indebted to Dr. M. A. Radcliffe-Taylor for advice in the preparation of this paper.

### References.

(1) W. R. Gowers: "Lumbago: Its Lessons and Analogues", *British Medical Journal*, Volume I, 1904, page 117.
(2) W. E. Dandy: "Treatment of Recurring Attacks of Low Backache without Sciatica", *The Journal of the American Medical Association*, Volume CXXV, 1944, page 1175.
(3) J. A. Key: "Intervertebral Disk Lesions are the Most Common Cause of Low Back Pain with or without Sciatica", *Annals of Surgery*, Volume CXXI, 1945, page 534.
(4) J. H. Young: "Intervertebral Disk Disease: New Syndromes and New Concepts", The Medical Journal of Australia, Volume II, 1945, page 234.
(5) B. H. Burns and R. H. Young: "Protrusion of Intervertebral Disk", *The Lancet*, Volume II, 1945, page 424.
(6) J. Cyriax: "Lumbago: Mechanism of Dural Pain", *The Lancet*, Volume II, 1945, page 427.
(7) F. Wood Jones: "The Principles of Anatomy as Seen in the Hand", 1944, page 214.
(8) F. A. Elliott: "Tender Muscles in Sciatica: Electromyographic Studies", *The Lancet*, Volume I, 1944, page 47.
(9) O. A. Beadle: "The Intervertebral Discs: Observations on their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities", Medical Research Council of the Privy Council, Special Report Series Number 161, 1931.
(10) W. E. Dandy: "Concealed Ruptured Intervertebral Disks: A Plea for the Elimination of Contrast Media in Diagnosis", *The Journal of the American Medical Association*, Volume CXVII, 1941, page 821.
(11) W. G. Deucher and J. G. Love: "Pathologic Aspects of Posterior Protrusions of the Intervertebral Disks", *Archives of Pathology*, Volume XXVII, 1939, page 201.
(12) J. H. Young: "Recent Advances in the Diagnosis and Treatment of Lumbar Intervertebral Disk Disease", The Medical Journal of Australia, Volume I, 1946, page 45.

## Reports of Cases.

### SPINAL ANÆSTHESIA AND THE PREGNANT WOMAN.

By Arthur E. Brown,
*Colac, Victoria.*

### Clinical Record.

On September 24, 1945, a woman, aged twenty-nine years, was brought to the operating theatre in the Colac Hospital for the purpose of undergoing the operation of Cæsarean section for the delivery of her seventh child. The expected date of confinement was October 1, and she was not in labour. The operation had been deliberately planned to deal with a persistent transverse presentation. Her health had been good throughout her pregnancy; her blood pressure was 130 millimetres of mercury, systolic, and 70 millimetres, diastolic, and her urine was free from albumin. At 8.15 on the morning of the operation she received by hypodermic injection one-quarter of a grain of morphine, and 1/100 grain of hyoscine, and she was taken to the operating theatre at 9 a.m. She was then not deeply narcotized by the morphine, and when at 9.6 a.m. she was placed in a sitting position for the spinal injection, she flinched at the needle prick. Two millilitres of "Percaine", 0·5% strength in isobaric solution, were injected into her spinal theca through the third lumbar intervertebral space, without difficulty, and without barbotage. This injection was immediately followed by a hypodermic injection of 0·5 grain of ephedrine, and she was laid flat again at 9.8 a.m. Five minutes later she retched slightly and her head was turned to the right side, but no vomitus escaped, and there was no evidence that any had emerged from her stomach. At 9.18 a.m., while her abdomen was being draped, her condition was good, and she moved her left arm about restlessly, so that the attendant sister took it in her hand to control it. At

9.20 a.m. there was a sudden change in her condition. Her skin became mottled and her colour bluish, and she ceased breathing. It is difficult to be certain whether her pulse ceased at the same time, but a few moments later it was imperceptible. Measures to retrieve her respiration included the passage of airway into the throat, the blowing in of "Carbogen" under light pressure, the intramuscular injection of 2·0 millilitres of "Coramine", artificial respiration, and an intracardiac injection of adrenaline; but no success was obtained, and her death was evident. The fœtal movements were present at the time of the cessation of respiration, but ceased during the attempts to revive the mother.

At the post-mortem examination, which was not carried out by a trained pathologist, it was established that all the organs examined, including the heart and lungs, were grossly normal, and there was no obstruction in the upper air passages by vomitus or any other foreign material. The death was apparently due to sudden respiratory failure; but the cause of this is still to seek.

### Discussion.

C. McIntosh Marshall, in his monograph "Cæsarean Section: The Lower Segment Operation", goes well into the question of the use of spinal anæsthesia in these operations, and after setting forth its various advantages he discusses (page 64) "the peculiar susceptibility to spinal anæsthesia of women undergoing Cæsarean section". He quotes Preissecker as making the following statement:

> It must nevertheless be admitted that the percentage of fatalities in pregnant women is higher than in non-gravid patients—indeed, as a result of frequent and unpleasant occurrences, the use of spinal anæsthesia has in many places been entirely given up.

Marshall adds that a number of other authors have stressed the fact that "the pregnant state renders the woman particularly sensitive to the effects of spinal anæsthesia"—as, for instance: "The combined experiences of Sussman, von Jaschke and Mayer embraced some 12,000 anæsthetics; there were four deaths, all in Cæsarean sections."

In attempting to explain this phenomenon Marshall states that such supersensitivity towards the drugs used in spinal anæsthesia has been accounted for in many ways.

> 1. The threshold of permeability of the capillary endothelium is lowered during pregnancy. Preissecker considers that this favours the entry into the circulation of substances injected intrathecally.
>
> 2. Von Jaschke saw a connexion between the existing lipoidæmia and the increased toxicity of spinal anæsthetic solutions during pregnancy (Franken).
>
> 3. The volume of blood diverted to the generative organs in pregnancy is greatly increased. The action of spinal anæsthesia is to shift a further quantity into the splanchnic circulation and the lower limbs; the venous return is thus diminished, the stroke volume of the heart reduced, and the vital centres are exposed to the dangers of anoxæmia. Shitosuma, Waters and Leavitt, Dieter and quite a number of others hold that it is mainly in this indirect way that respiratory and vasomotor paralysis is brought about in the majority of those cases which show symptoms of collapse or terminate fatally.
>
> 4. Pankow some years ago expressed the view that the presence of labour pains was inimical to the safe working of spinal anæsthesia. Franken is now the chief exponent of this belief, and explains their unfavourable effects in the following way: Uterine contractions cause little change in the intrathecal pressure. He has, however, clearly demonstrated by manometric experiments that the pressure of the cerebro-spinal fluid is considerably raised when the pains of labour are associated with crying out, marked restlessness, or bearing down efforts. Now Hartwich has proved that similar efforts or emotional disturbances in children bring about similar pressure changes; as a result solutions injected into the lumbar canal were rapidly translated to and could be readily recovered from the *cisterna magna*. Proceeding by analogy Franken supposes that this is what occurs in the labouring woman.

The fourth of these hypotheses can, I believe, be ruled out entirely as causing death in the present case. Labour had not set in, and could not be a factor. Further, the respiratory paralysis could hardly be due to an unusual ascent of the "Percaine" in the spinal theca, because twelve minutes after the injection, and only two minutes before the respiratory failure, the patient was exhibiting decided evidence that the motor centres in the cord as low down as the first thoracic segment were entirely unaffected by the drug.

K. B. Heard[1] remarks that "it is frequently stated that the possibility of excessively high anæsthesia is greater in pregnancy than in normal health, because of altered dynamics of intraabdominal pressure and circulation", and he quotes his own series of 8,472 operations of all sorts under spinal anæsthesia, among which intercostal paralysis and respiratory embarrassment occurred ten times. This gives an incidence of intercostal failure of 1·2 per 1,000, or 0·12%. Included among these 8,472 operations were 279 Cæsarean sections, and in these intercostal failure occurred twice, making an incidence in this restricted series of 0·7%. Despite the apparent significance of his figures, Heard believes that the risk of spinal anæsthesia is no greater in pregnancy than in any other condition, and he is emphatic that spinal anæsthesia is the best method to use in such operations as appendicectomy, when they become necessary during pregnancy.

I cannot believe that the patient whose case I have here reported died from intercostal paralysis. The complete and dramatic suddenness of the change from comparatively good condition to an evidently hopeless state would appear to me to be evidence of central respiratory rather than intercostal paralysis.

I do not believe that the preliminary injection of morphine and hyoscine was an important factor in the death of my patient. It is my belief that in the use of spinal anæsthesia generally such preliminary medication is an important adjunct, both for the purpose of avoiding the emotional element in producing severe falls in the blood pressure, and also to reduce resistance to the anæsthetizing action of the "Percaine" itself. To healthy adults I usually give a larger dose than this woman received. After receiving one-quarter of a grain of morphine and 1/100 grain of hyoscine ninety minutes prior to operation, they also receive one-sixth of a grain of morphine and 1/150 grain of hyoscine thirty minutes before operation. I have never found such dosage to cause any ill effects whatever in the normal adult. When given to elderly men with prostatic disorder, the routine dosage does in many instances produce such depression of vital activity to set up temporary oliguria, even at times almost anuria; this, however, has always passed off harmlessly. To these patients I give a deliberately reduced dose.

I have always feared the possible effect on the baby of administration of morphine to the mother before Cæsarean section, and mainly for that reason I have used spinal anæsthesia very little in Cæsarean section operations. I am therefore unable to quote any useful personal figures as to risks of its use in my own practice. My total experience of spinal anæsthesia has been approximately 1,000 cases; but, including the case here reported, I have used it in only seven Cæsarean sections. I have never met with any happening to cause me the slightest anxiety in any other case. Such a fatality, however, when it does occur, is so dramatic and so harassing to one's sense of responsibility that I find myself quite unable to act on Marshall's reassuring summing up:

> Comparable statistics showing the results of spinal and general anæsthesia in the lower segment operation are few. They do, however, support the impression that despite the occasional death which must inevitably result from its use, spinal anæsthesia will yield better total results than the ordinary inhalation method.

Marshall then publishes tables of comparable statistics from three series of Cæsarean sections performed under general anæsthesia, including his own from Liverpool. The total cases in the three series number 1,180, and the percentage mortality rates in the three were 3·3, 3·8 and 2·2 respectively. Similar figures from four series of

Cæsarean sections performed under spinal anæsthesia, also including the Liverpool series, give a total of 662 operations. The percentage mortality rates in these four series were respectively 1·5, 1·3, 1·4 and 1·6. In commenting on these striking figures Marshall makes the following statement:

> Fatalities from spinal anæsthesia are sudden and dramatic, and the relation between cause and effect is so obvious that, excepting only air embolism, they cannot possibly be confused with any other form of death. On the other hand there is no such narrow connexion between general anæsthesia and the dangers to which it can give rise.

Death from "shock", "cardiac failure", "uterine atony" or delayed or reactionary hæmorrhage are those which may be connected with anæsthesia by the inhalation method, and this explains the lower total mortality rate under spinal anæsthesia.

If this is so, it would seem that Cæsarean section must carry its own enhanced dangers for inhalation anæsthesia, which deserve close inquiry. For if, after allowance is made for the admittedly increased susceptibility of pregnant women to the dangers of spinal anæsthesia, the mortality rate following the use of the inhalation method is approximately twice as great, a state of things would seem to exist which is not found in conditions other than Cæsarean section. It may be added, in this connexion, that Marshall states definitely that the spinal anæsthetic series of the Liverpool Maternity Hospital, quoted above, included the great majority of those patients who for one reason or another were regarded as "poor risks".

Bourne and Williams[2] are most decided about the risks of using spinal anæsthesia for Cæsarean section:

> There is a mortality risk attached to spinal anæsthesia which appears to be unavoidable. In a letter to the B.M.J. (Oct. 9th, 1937) the late Dr. Herbert Spencer drew attention to the fact that many authorities, including De Lee, Krönig, Brouha, Voron, Winter and Halban, regarded spinal anæsthesia as dangerous in pregnancy and in labour perhaps because of a special bulbar sensibility of these women to the action of cocaine compounds.

Bourne and Williams note that various spinal anæsthetic preparations which are synthetic substances, and not cocaine derivatives, have produced plenty of fatalities. And they contrast the successful lists published by Ballen and Rufus Thomas with the disastrous experiences of Pla and Ricci, who, in 45 Cæsarean sections, had three maternal deaths and three "near deaths" under spinal anæsthesia. The great contrast between the numerous large satisfactory series and the equally numerous tragic series makes one wonder what if any influence is exercised by differences in technique employed by the various workers in producing these dramatically different results.

That the question of the special dangers of spinal anæsthesia in pregnant women is not yet settled in the minds of doctors generally is shown by the fact that, in spite of the publication of these grave warnings in widely read books and journals, there has recently been a large correspondence in the *British Medical Journal* on the subject of the use of spinal anæsthesia in obstetrics and for Cæsarean section, in which the majority of writers have seemed to hold it to be the ideal anæsthesia for the purpose, making but little mention of any exceptional risks involved or of grim personal experiences. It will be most interesting to note whether in the future a number of tragedies will follow its use, which will demonstrate beyond doubt that pregnant patients do bear that special risk which Marshall's quotations and those of other writers seem to indicate so clearly, and which my own experience has impressed upon me, or whether with improving technique generally it will be found that it was technique rather than an inherent susceptibility which has been at fault in the past.

Another direction in which inquiry might be directed with great profit would be the further analysis of such large series of cases as those recently published by Corlette.[3] He uses these series to demonstrate the large percentage of deaths due to spinal anæsthesia generally, in comparison even with the results from chloroform anæsthesia in the heyday of that drug. It is probably not possible now to determine how many of the spinal anæsthetics administered in those series were administered to pregnant women, or to determine how different the mortality rates would appear were cases of pregnancy excluded from the series; but if it was possible, such an inquiry would be both instructive and important. In the series quoted by Marshall of Sussman, von Jaschke and Mayer, for instance, there appear 12,000 cases of spinal anæsthesia with four deaths. But all these four deaths were in cases of Cæsarean section, and if all these special risk cases were excluded, the series would probably show some 10,000 or 11,000 cases without a death. My own figures also at present show approximately 1,000 cases of spinal anæsthesia with one death; but if my seven Cæsarean sections were excluded, the result would be approximately 1,000 cases without fatality. In its relation to Corlette's paper this point is important. Spinal anæsthesia has so many and such definite advantages over ether, both for the surgeon and for the patient, that it would be as grave an error to overrate its dangers as it would manifestly be to underrate them.

### References.

(1) K. B. Heard: "Anesthesia in Cæsarean Section", *Surgery, Gynecology and Obstetrics*, Volume LXX, 1940, page 657.
(2) A. Bourne and L. H. Williams: "Recent Advances in Obstetrics and Gynæcology", 1945.
(3) C. E. Corlette: "Spinal Anæsthesia and Chloroform: A Comparison of Mortality", THE MEDICAL JOURNAL OF AUSTRALIA, April 20, 1946, page 545.

## A CASE OF EXOMPHALOS SUCCESSFULLY TREATED BY EARLY OPERATION.

By GEORGE R. HEMMING,
*Medical Officer, Colonial War Memorial Hospital, Suva, Fiji.*

### Clinical Record.

A FULL-TERM male Fijian child was delivered with considerable difficulty owing to some obstructive factor following the normal birth of the head. When the delivery was completed a large hernia into the umbilical cord was found, containing practically the whole of the small intestine, part of the large intestine and the lower pole of the right lobe of the liver. The sac was made up of a fine, translucent membrane, the amnion, and the contents of the abdomen could be seen clearly through it. The base of the sac was about two and a quarter inches in diameter. The child looked normal otherwise; it was not weighed at birth.

Operation was performed about forty-five minutes after birth, a spirit pad having been placed over the sac in the meantime. The abdominal wall was infiltrated round the sac with a 0·5% solution of "Percaine" and the sac was excised about one-quarter of an inch from the skin margin. Bleeding points, which were chiefly in the lower part of the sac, were secured with artery forceps and tied off with fine catgut. The small and large gut was intensely injected, having the appearance of thick red velvet, presumably from the trauma involved in the delivery. There was a quantity of free blood-stained fluid in the abdominal cavity. It was now found to be impossible to return the stomach and gut to the abdominal cavity, and general anæsthesia was induced with ethyl chloride followed by ether. The gut was finally returned with difficulty, and a large number of silkworm gut sutures were inserted, including all layers of the abdominal wall. No attempt was made to freshen the skin edges, the line of incision of the sac being about one-quarter of an inch distant from the skin. The immediate post-operative condition appeared satisfactory.

The child was extremely ill for several days, and it is probably worth while recording some details of the measures taken to feed the child and combat dehydration (Table I).

By this time the child was obviously recovering and was taking some feeds from the breast. On August 29 the weight was approximately eight pounds ten ounces.

TABLE I.

| Date. | Fluid Given. | Amount of Vomitus. | Bowel Actions. | Notes. |
|---|---|---|---|---|
| 19.8.45 .. .. | "Karilac" solution, 8·7 ounces. | 3·3 ounces. | 2 | Sulphathiazole, 0·1 gramme every four hours; general condition satisfactory. |
| 20.8.45 .. .. | "Karilac" and colostrum, 12·5 ounces. | 4·5 ounces. | 8 | Sulphathiazole, 0·1 gramme every six hours. Tap water *per rectum*, not retained. |
| 21.8.45 .. .. | "Karilac" and colostrum, 13·5 ounces; saline solution subcutaneously, 1·0 ounce; fluid *per rectum*, 1·0 ounce. | 4·0 ounces; stomach aspirated; 2·5 ounces. | 4 | Sulphathiazole, 0·1 gramme every six hours. Condition fair. Jaundice pronounced. Rapid loss of condition. |
| 22.8.45 .. .. | "Karilac" and breast milk, 8·5 ounces; saline solution subcutaneously, 4·0 ounces; fluid *per rectum*, 2·0 ounces. | Nil; stomach aspirated, 5·4 ounces. | 10 | Sulphathiazole, 0·1 gramme every six hours. Temperature 103° F., pulse rate 160 per minute. Child considerably wasted. |
| 23.8.45 .. .. | Expressed breast milk, 9·0 ounces. | 3·0 ounces. | 10 | Sulphathiazole, 0·1 gramme every six hours. Temperature 100·6° F., pulse rate 148 per minute. |
| 24.8.45 .. .. | Expressed breast milk, 12·0 ounces. | —— | 4 | Sulphathiazole continued. Temperature 100·8° F. Suture line inspected; some healing. |
| 25.8.45 .. .. | Expressed breast milk, 10·5 ounces. | —— | 1 after an enema | Sulphathiazole. Temperature 99° F. Some sutures removed. |

When all sutures were removed a raw area the size of a florin was present; this was dressed with eusol and later with flavine emulsion, and later still with scarlet ointment. This raw area gradually became epithelialized and the abdominal wall was supported with "Elastoplast" strapping and on the child's discharge from hospital with a soft corset.

The mother and child were discharged from hospital on September 25 and the child has progressed very well since. There is some weakness of the abdominal wall, but no actual hernia. His weight on December 21 was fourteen pounds seven ounces, and his general condition good.

### Discussion.

The condition is not common, and I have not previously had occasion to operate for it. This is the first case in 2,035 deliveries amongst Fijians and Indians since I have been associated with this hospital.

The only reference I can find is a case reported by Leonard Ley in *The British Journal of Surgery* of October, 1942, at page 168; Bailey and Love also refer to the subject in "A Short Practice of Surgery".

### Acknowledgements.

I wish to thank Dr. H. S. Evans, Acting Director of Medical Services, Fiji, for permission to publish this case.

## FIBROMYOMA OF THE SMALL INTESTINE CAUSING INTUSSUSCEPTION.

By J. M. O'CONNOR,
*Brisbane.*

### Clinical Record.

MRS. E.I., aged thirty-four years, presented herself for examination on January 3, 1946. She complained of attacks of abdominal pain during the previous three years. The attacks were increasing in frequency and severity. She had had her appendix removed two years previously. The pain usually commenced in the middle or upper part of the abdomen and radiated indefinitely. Vomiting was rare and there was no history of abnormal bowel function, of dysuria or of hæmaturia. The menstrual function was normal. The pain commenced and ceased suddenly, and the patient was well between attacks.

Examination of the patient revealed that her appendicectomy had been performed through a lower mid-line incision. The only abnormality which could be discovered was some slight tenderness under the right costal margin. Cholecystography revealed a normal gall-bladder. X-ray examination of the stomach and duodenum after a barium meal disclosed that up to six hours none of the barium had passed through the pylorus; there was an obstructive lesion involving the pylorus and duodenum. This was thought most probably to be a cicatrizing pyloro-duodenal ulcer.

The patient was then referred to Dr. C. A. Thelander for gastro-jejunostomy. However, after he had examined her, Dr. Thelander said that he did not agree with the X-ray findings and that he considered the delay in the emptying time of the stomach to be due to spasm of the pylorus of a reflex nature and "secondary to some intestinal lesion, such as a recurring volvulus, intussusception, or something of the sort". He suggested laparotomy for investigation.

Laparotomy was performed by Dr. Thelander on March 29. At operation the pylorus and duodenum were found to be normal. There were many adhesions from her previous operation, and some of these were divided so as to give access to the lower part of the abdomen. Eventually an intussusception of about the middle portion of the ileum was delivered into the wound. It was reduced to the apex, which was found to be solid and irreducible. Consequently this segment was resected and an end-to-end anastomosis was performed. Recovery was uneventful and the patient left hospital on April 16.

Dr. G. C. Taylor, pathologist, reported as follows on the segment of ileum which was removed: "Growth in small intestine is a fibromyoma, about four inches in length, covered by a congested mucous membrane, and showing areas of fatty degeneration. No evidence of malignancy seen."

According to all authorities, fibromyoma of the ileum is a rare condition. It would appear that three types have been described. The first grows from the longitudinal layer of smooth muscle towards the lumen of the bowel, pushing the mucous membrane before it. The case under discussion was considered to be of this, the commonest, variety. The second type grows from the longitudinal smooth muscle subserously and forms a subperitoneal tumour. The third type grows from the circular layer of smooth muscle, stays in that layer, and forms a ring tumour around the bowel.

Nothnagel's "Diseases of the Intestine and Peritoneum" has the following interesting paragraph: "In carcinoma of the intestine the formation of an intussusception must be considered an exceptional occurrence. In benign tumours of the intestine, however (adenoma, fibroma, myoma, lipoma), in the superficial, particularly in the pedunculated

forms—so-called polypi—intussusception occurs with great frequency. When it is remembered how rare, absolutely speaking, these benign tumours of the intestine are, it is surprising in what a large percentage of these cases invagination of the bowel occurs."

# Reviews.

## A POCKET SURGERY.

The first edition of "A Pocket Surgery" by Mitchener and Whyte was published only three years ago, and already the second edition is to hand.[1] The book was written originally to supply the needs of young doctors whose surroundings were unsuited to much study because of the war. It was a volume which could readily be carried by a regimental medical officer in his kit. Now that the war is over, some effort has been made to adapt the book to peace-time requirements. It would have been better to have accepted the inevitable fact that the volume is no longer required.

It is hard to say to whom the book will be of use. From what can be seen of the keenness of army doctors undergoing rehabilitation the tendency is for them to read too widely rather than to be content with a smattering of surgical knowledge. Nor is the book intended for the use of students whose examination answers need to contain greater detail than can be found here.

For the sake of space the style has been made compact to the point of crudeness and, frequently, loss of clarity. This is regrettable. One must also deplore the omission of reasons for the various forms of treatment. For example, the intravenous therapy suggested for burns is described thus: "Transfuse serum, plasma, or whole blood (NEVER saline—œdema of lungs)." This is quite useless advice to the uninitiated, apart from being open to question. Extradural hæmorrhage still receives twice the attention that is devoted to subdural hæmorrhage. The signs and symptoms of the latter are covered by the illuminating sentence: "Symptoms are variable."

It must be pointed out that the book contains much pithy common sense; the main criticism is that its publication was unnecessary.

## CLINICAL NEUROLOGY.

In his "Introduction to Clinical Neurology", Dr. Gordon Holmes has given us a simple and straightforward exposition of the facts concerning structure and functions of the nervous system which are necessary for an adequate appreciation of symptoms and signs.[2] Further, the methods of examination described are strictly clinical without reference to the use of X rays or electroencephalography. Nor do we find any mention of changes in the physical and chemical properties of the cerebro-spinal fluid. The short chapter on pathology deals with the effects of rapid and gradual lesions and with the characteristics of diffuse and systemic diseases of the nervous system. The leading functions such as movement, sensation, coordination, equilibrium and speech are covered in that systematic and logical manner which has earned Dr. Holmes a reputation as an exceptional teacher. In a useful chapter on the mental state the attitudes of the patients with psychoneurotic and organic conditions to their respective symptoms are contrasted in a helpful manner. The functions of the hypothalamus are mentioned in the section on the autonomic system which deals mainly with vasomotor and pilomotor reactions. Thirty-six simple diagrams are distributed through the text which contains few proper names and no references. This is not a book for those who seek after signs, and the experienced neurologist may find little that he does not already know. But those who have had the privilege of seeing Dr. Holmes at work and who read this book carefully will appreciate how much can be learned from the intelligent application of comparatively simple clinical methods, though the simplicity of the author's style might appear to conceal the immense experience which lies behind his words. The book is essentially practical and no space is wasted on speculation. As an introduction to more advanced studies and to the practice of neurology, the student, pre-graduate or post-graduate, will find the work indispensable.

[1] "A Pocket Surgery", by Philip H. Mitchener, C.B., C.B.E., T.D., M.D., M.S. (London), F.R.C.S. (England), D.Ch.Hon. (Durham), D.L., and A. Hedley Whyte, D.S.O., T.D., M.B., M.S. (Durham), F.R.C.S. (England and Edinburgh); Second Edition; 1946. London: J. and A. Churchill, Limited. 7¼" × 4¾", pp. 280. Price: 8s. 6d.

[2] "Introduction to Clinical Neurology", by Gordon Holmes, M.D., F.R.S.; 1946. Edinburgh: E. and S. Livingstone Limited. 9½" × 6", pp. 192, with illustrations. Price: 12s. 6d.

## THE CAUSATION OF APPENDICITIS.

Before a writer expends time and energy in an attempt to explain alleged variations in the frequency of a disease, he must be very sure of the facts on which he bases his hypothesis. A. Rendle Short, in his small book on "The Causation of Appendicitis", accepts as a fact that there was a very great increase in the incidence of appendicitis in the United Kingdom between 1895 and 1905, and then proceeds to search for a plausible explanation for this increase.[1] To an unbiased critic the evidence of this increase is far from satisfactory.

It is certainly true that if one compares the decade preceding 1890 with that preceding 1910, there was a huge increase in the number of patients entering hospital with this diagnosis, in the operation of appendicectomy, and in deaths classified as due to appendicitis. But a study of the literature on appendicitis prior to 1890 will show that only peritonitis or abscess due to appendicitis was ever recognized; it was not deemed possible to diagnose the disease prior to perforation and the development of these sequelæ. Obviously then the incidence of this disease would seem vastly lower than in the decade prior to 1910, when not only was the early recognition of uncomplicated appendicitis understood, but the appendix was also removed constantly for a finding of chronic tenderness at McBurney's point, a condition which is now recognized as being a psychosomatic phenomenon—part of an emotionally induced neuralgia. In a similar way the mortality rate of appendicitis increased with the increase in appendicectomy, and need bear no direct relation to an increase in appendicitis.

To an author like Rendle Short, who states that "quite 40% of patients with acute appendicitis not operated on would have a long and serious illness, many would die", such a consideration might not appeal; but if he would consider that the rise in the mortality figures in England coincided with the developing practice of operating in the acute stage, and not as a prophylactic measure after the attack had subsided, he might see some force in the argument.

The conclusion is inescapable that, whether there was a true rise of the disease at all between 1895 and 1905 in the United Kingdom, it was vastly less than the figures for appendicectomy would suggest, and that the increase in deaths regarded as due to the disease was due to replacement of simple sedation for "colic and wind", "plague in the guts", "stoppage of the stomach", or what not, by ill-advised surgery (because of technical weaknesses or at an improper phase of the disease) and its equally ill-advised accompaniment of purgation. In those places where wiser methods of treatment have again given place to the *"furor operandi"*, appendicitis is rapidly returning to its proper position as a relatively uncommon and often trivial complaint.

Accepting the fact of a great increase in the disease, Rendle Short next investigates the possible changes in the dietary of the United Kingdom that might be held responsible, and concludes that "appendicitis is due to the replacement of cellulose-containing foods by others, and especially by white bread and more meat". He has not considered, however, the implication of the fact that an equally great increase in appendicectomy occurred in Australia and in rural parts of the United States, where no such dietary change can be postulated. Nevertheless, here he is possibly on firmer ground, and medical men who have had the opportunity of observing the increased incidence of appendicitis in native races who have changed in a generation from such a diet to that of the white races will support him in the belief that the present diet of civilized man conduces to appendicitis as well as to other diseases of the gastro-intestinal tract.

To the extent that such a change occurred in the period under review, Rendle Short may be right in insisting on an increase in appendicitis, but whether this justifies the writing of this book, and what the civilized world is to do about it, remain matters for conjecture.

[1] "The Causation of Appendicitis", by A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; 1946. Bristol: John Wright and Sons Limited: London: Simpkin Marshall (1941) Limited. 7¾" × 5¾", pp. 88. Price: 10s.

The Medical Journal of Australia

SATURDAY, OCTOBER 5, 1946.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## THE NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL.

Mention is made in these pages from time to time of the meetings of the National Health and Medical Research Council, that dicephalous creation of the Commonwealth which even to this day is not understood as it should be by the practising members of the medical profession. For some considerable time attempts have been made from several directions to separate the research activities of the council from those dealing with national health, and for a while hopes ran high that these efforts would be successful. Though it does not seem likely that anything of the kind will be done in the immediate future, there is no reason to give up hope. In the meantime the medical profession, as we have insisted on previous occasions, must recognize that the council, with its present organization and with the restricted funds at its disposal, has done a good deal of useful work. The twenty-first session of the council was held at Canberra on May 28 and 29, and the report of the meeting has now been published. Some of the matters dealt with at that meeting should be enumerated and many of them will be seen to have far-reaching possibilities. At the last meeting, medical research was quite overshadowed by questions of national health, but this is probably explained by the fact that grants for research are not as a rule made in the middle of the year.

On the research side the constitution of the Reference Subcommittee was discussed. This is the committee which considers applications for research grants and makes recommendations to the full council. The Acting Chairman of the council stated that a good deal of criticism had taken place on the ground that workers actually engaged in research were not adequately represented on the subcommittee. Considerable discussion ensued and it was eventually decided that the Reference Subcommittee should be disbanded and that a new committee to be known as the Medical Research Advisory Committee should be formed with the following members: Professor H. R. Dew (chairman), Dr. H. J. Ritchie, Dr. W. F. Simmons, Professor M. L. Mitchell, the Commonwealth Director-General of Health, Professor F. M. Burnet, Professor H. K. Ward, Professor R. D. Wright, Dr. E. V. Keogh. The first five are members of the National Health and Medical Research Council. The appointment of the last four as members of this advisory committee will give general satisfaction. Dr. R. E. Richards is to be medical recorder of the new committee.

On the public health side mention must first be made of the field of nutrition. The council decided at the instance of its Nutrition Committee to publish as one of its special report series a report entitled: "Tables of Composition of Australian Foods". This report has been prepared under the direction of the Nutrition Committee by Miss A. Ormond, B.Sc., and is regarded as likely to meet full Australian requirements. The only material of the kind available at present is a report of the Medical Research Council of Great Britain, "Composition of Foods in England", and a report issued by the Australian Council for Scientific and Industrial Research on the composition of some Australian foods. At a previous session the council had considered the very difficult question of the introduction of legislation to control claims by advertisers of the vitamin and mineral value of foodstuffs. State representatives had been asked to consider the matter. Several State representatives pointed out that great difficulties would arise in the policing of any regulations that might be introduced. It will at once be obvious that though the mineral content of a proprietary preparation may be determined without much difficulty, the estimation of its vitamin content would involve a long and complicated investigation. Even if the exact composition of a foodstuff was determined, there would be no certainty that every subsequent batch of samples would have a similar composition. This is a matter in which the known probity and general commercial reputation of a manufacturer must be taken into account by those who have to give an *imprimatur* to a commercial preparation used as a foodstuff. It might be possible to establish an investigational bureau which would buy on the open market products thought to be suspect and subject them to searching examination. If deception by a manufacturer was discovered in this way, he should be debarred from all future operations on the market of medical foodstuffs. Professor D. H. K. Lee, of Queensland, submitted to the council a special report on human climatology, based on information obtained by him overseas; he made recommendations on the application of the findings to Australian problems. Though the council considered the report, it decided to do nothing about it for the present. In the matter of tuberculosis, the council considered a communication by Dr. Cotter Harvey on the results obtained with "B.C.G." vaccine in Canada, on the advisability of using the vaccine in Australia, and on the possibility of its manufacture by the Commonwealth Serum Laboratories. Dr. F. G. Morgan, Director of the Commonwealth Serum Laboratories, who was present as one of the Commonwealth representatives, said that the vaccine could be made at the Serum Laboratories, but that it would be necessary for a new strain to be obtained from overseas and developed as a special product. The council referred the question of the use of the vaccine to its own Committee on Tuberculosis for consideration and report at its next meeting. Mr. N. E. Murray addressed the council on the future scope of work at the Acoustic Laboratory. It will be remembered that Mr. Murray, with Dr. G. Reid,

contributed an article to this journal on the work of the laboratory in the issue of May 4, 1946. The council showed its wisdom by inviting members of the Schools Medical Officers' Conference, which was meeting at Canberra, to hear Mr. Murray's address. The subject was discussed at some length and the council then resolved that the Commonwealth should set up an organization to be known as the Commonwealth Acoustic Laboratory, and controlled by the Commonwealth Department of Health, under conditions similar to those operating with regard to the Commonwealth X-Ray and Radium Laboratory. It also expressed the opinion that, as the laboratory would be concerned with acoustic standards and industrial hygiene activities and with work for repatriation and defence services, the cost should not be a charge against the council's future research vote. At the instance of the Victorian Branch of the British Medical Association, Dr. H. N. Featonby raised the question of prophylaxis against endemic goitre in Gippsland. It was suggested that a survey of the iodine content of water and soils throughout the area should be made. Dr. F. W. Clements pointed out that no rural area in Australia had been covered by a food consumption survey. He thought that it would be possible to conduct a food consumption survey in conjunction with the goitre survey that had been requested. It was decided that the survey as outlined by Dr. Clements should be carried out and that the cost should be borne by the departmental nutrition vote.

The public health questions that have been mentioned are of the greatest importance; others were discussed. Among these it seems a little quaint to read that the Commonwealth Treasurer had approved that "a subsidy for venereal diseases" should be paid to two States. Two matters remain to which attention should be drawn. The council received from the Acting Prime Minister a communication that he had received from the Premier of Western Australia regarding the proposed development of a medical school in Western Australia. The council expressed the opinion that the development of a medical school in Western Australia was necessary in the best interests of medical practice and medical research. This is a matter which has been discussed in Western Australia for some considerable time. As a matter of fact Professor P. MacCallum, of the University of Melbourne, has gone to Perth at the request of the Government of Western Australia to give advice on the subject. He has already pointed out that there must be no tinkering with the establishment of a medical school in Perth, that a medical school must be more than a training ground for people who wish to obtain a degree that will enable them to earn a living. It must, he declares, be more than a glorified technical school. It must be a centre of medical thought and development, a source of inspiration and a centre of research. It will also be costly. The resolution of the National Health and Medical Research Council will, it is to be hoped, stimulate the Western Australian Government to accept Professor MacCallum's advice.

The last matter to be mentioned is one to which full approval must be given. At an earlier meeting of the council a discussion took place in regard to post-graduate study tours abroad for medical officers who are permanently employed by governments or public bodies. It was resolved that conditions in regard to these tours should be formulated and sent to interested departments. Applications will be invited and considered at the next meeting. It is to be hoped that those who are sent will be chosen from officers who are not too set in their ideas, who, to quote Professor Walter Murdoch, have not closed their ledgers.

# Current Comment.

## BERIBERI HEART DISEASE.

The great humanitarian organizations of the nations are concerned with serious problems of malnutrition, and recent experiences in prison camps have also given the medical world striking and terrible reminders of the slender margin of safety which there is in the world's dietary. The risk of deficiency disease is present, however, not only in war devastated zones; it is always with us in one form or another. M. A. Blankenhorn, C. F. Vilter and R. S. Austin have collected a series of twelve cases of beriberi heart disease, seen in the Cincinatti General Hospital.[1] Though they acknowledge the early recognition of beriberi in its circulatory manifestations by workers in the tropics, and particularly the contributions of Wenckebach, most of the literature quoted by them in this article is derived from comparatively recent American sources. It now seems to be well established that the original criteria have to be modified. For example, rapid action of the heart is not always observed, and the predilection of the process for the right side of the heart as described by Wenckebach is not borne out by modern experience. Perhaps, too, the descriptions of the wet form of beriberi have obscured the true picture, or at least a picture frequently seen. Among 12,000 admissions to hospital, twelve proven cases are described. This incidence is low, but the important point is that if the part played by the $B_1$ vitamin complex is realized in those cases in which it is significant, a specific and valuable form of treatment is at hand. Blankenhorn has previously laid down criteria for the diagnosis of beriberi heart disease, and maintains that these are much more in keeping with present-day experience. These criteria are the insufficiency of evidence of other ætiology, a history of a thiamin-insufficient diet for a period of several months, signs of neuritis or pellagra, enlargement of the heart with sinus rhythm, œdema of the dependent or gravitational type, raised venous pressure, and recovery with decrease in the size of the heart. To these may be added the presence of minor electrocardiographic changes, but the cautious cardiologist will probably agree that these will not make up his mind for him. In the event of death, the autopsy findings should be consistent with the known pathology. The important findings are overall hypertrophy or dilatation, hydropic degeneration of the heart muscle, and the conduction system, and some intercellular œdema. Five of the authors' patients died in hospital, and autopsy was performed in three cases. Some interesting findings are recorded in the central nervous system, but the details of these cannot be discussed here at present, though it is of interest to note that changes were found in both the general peripheral and the autonomic systems.

The symptoms of the patients were in general those of degenerative heart disease, but careful inquiry into the history revealed the potential or actual deficiencies of their previous diets. The suspicion thus aroused was confirmed by the findings of neural or cutaneous lesions, and the response to treatment gave further support to the diagnosis. Large doses of thiamin were used by the intravenous route, and though significant decrease in the size of the heart was demonstrated in the successful cases, in only one instance was there dramatic improvement. Of course, as the authors point out, the changes in the heart have in such cases been going on for a long time, and such changes will inevitably reach the irreversible stage if efficient measures are not taken early. Even so, success

[1] *The Journal of the American Medical Association*, June 29, 1946.

must not always be expected, for the patients are usually middle-aged, and in this series, as is so often the case, they were all addicted to the excessive use of alcohol. The alcoholic factor is of peculiar importance; not only does it tend almost automatically to limit the intake of thiamin, and to disturb its balance with other elements of the diet, but its effects upon the digestive tract and allied mechanisms will lower absorption. The neurological signs observed were loss of deep reflexes, tenderness of the muscles, and sensory disturbances, though the latter are less reliable as evidence. The lesions of the skin were acute erythematous and chronic keratinized areas of the type seen in pellagra, and were usually accompanied by some degree of glossitis. It is perhaps significant that when death occurred it came rather suddenly as a rule. This is a subject worth further attention. Though nobody in this country should suffer from a substantial thiamin deficiency, such persons are still seen, and alcohol addiction is in this regard important. There is at the present time a lot of talk about vitamin *E* in heart disease, but here is a well-known vitamin deficiency which is the prime cause of recognizable heart disease and whose relief is based on much sounder premises.

## VINCENT'S INFECTION OF THE GINGIVÆ.

The condition known as Vincent's angina, or preferably Vincent's disease, is seen in the oral cavity and affects the tonsils, fauces and gums. Ulceration of the gums has been described as the most important manifestation of Vincent's disease. During the war of 1914-1918 this form of gingivitis often affected debilitated soldiers and was known as trench mouth. In this infection the predominating organism is the *Spirochæta vincenti*, and with it is found a long fusiform tapering bacillus, *Bacillus fusiformis* (*Fusiformis fusiformis*). Ulceration of the gums by Vincent's disease, also known as fusi-spirochætal pyorrhœa, is very common. Beattie and Dickson state in their textbook that in almost every case examined by them enormous numbers of the two organisms mentioned were found in varying proportions, along with leptothrix-like and mould-like filaments and mixed cocci and bacilli, including streptococci. Pyorrhœa is one of those conditions whose name is bandied about a good deal, but, like so many others of common repute or disrepute, it is not clearly understood. To gain an insight into its pathology is not, as a matter of fact, an easy thing to do. The ordinary textbooks of pathology devote but little space to it. For this reason attention should be directed to a recent study by A. Frank Stammers, who writes from the Dental Research Department of the University of Birmingham Medical School.[1]

This author's study is based on the results of his examination of 3,000 sections of gingival tissue taken from 21 patients suffering from Vincent's infection. Sections were taken from different parts of a mouth at the same time and from the same mouth at different times. All stages of the infection were covered, from the apparently simple to the acute fulminating ulcerative type. To avoid the distortion attendant on the use of local anæsthesia, tissue was removed with the patient under nitrous oxide and oxygen anæsthesia. Some of the terms used in the description of the sections must be defined. The term "oral epithelium" refers to that part of the mouth epithelium which covers the outer side of the gum as far as the gingival crest. The "crestal epithelium" is situated on the crest at the junction of the oral and "pocket epithelium". The last-mentioned is the epithelial lining from gingival crest to the bottom of the pocket; it is not to be confused with the epithelium of the "epithelial attachment", which extends from the bottom of the pocket for a variable distance towards the apex of the tooth.

The appearance of sections passing through an area of ulceration was found to be constant. One section, as an example, shows an ulcer which is still confined to the pocket side of the gum and extends to the gingival crest, which is being progressively undermined and destroyed. The oral epithelium retains its normal structure nearly to the crest—as far as it has healthy subepithelial connective tissue papillæ from which it can obtain nutriment. Only that part of the oral epithelium overlying inflamed connective tissue papillæ containing congested blood vessels shows signs of surface disintegration. The pocket epithelium has been completely destroyed and an ulcer extends deeply into the connective tissue of the gum. The deepest part of the ulcer is situated approximately midway between the crest and the bottom of the pocket, and is regarded as an indication of the site of the original breach of surface. A thick slough covers the ulcer, fills the pocket and extends over the gingival crest. Beneath the ulcer and walling it off from the deeper structures is a zone of intense inflammatory reaction. This inflammatory reaction was a constant feature in all sections. As will be seen when Stammers's views on treatment are stated, it is important to note his finding that numerous organisms, including fusiform bacilli and spirochætes, could always be demonstrated in the slough and superficial layers of living tissue, but no recognizable bacterial forms could be detected deep in the tissues, although many small particles, thought probably to be cell debris and fibrin, could be seen. This is not to say that no organisms were included in the deep tissues. Stammers quotes work by Pickworth showing that streptococci may grow in living but ischæmic tissue without active tissue reaction.

Stammers makes special reference to the part played by the connective tissue of the gum, pointing out that but scanty reference is made to it in the literature. He shows that not only do the proliferating processes of epithelium grow down into the connective tissue as a result of irritation, but that the connective tissue papillæ thrust outwards towards the surface of the pocket in order to bring the benefit of a free circulation nearer to the site of irritation. He regards the presence in many sections of a well-keratinized layer as proof that the nearer approach of the papillæ to the surface is not merely the result of erosion of the superficial epithelial cells. The gingival hyperæmic reaction appears to promote an excellent defence against threatened invasion of the tissue by organisms situated in the pocket, since it brings to the support of the overlying epithelium fresh body fluid with antibodies, leucocytes and additional powers of oxygenation. Stammers points out, however, that as a result of trauma hyperæmia of the gingivæ results sooner or later in rupture of some of the capillaries with hæmorrhage, thrombosis and congestion. The earliest hæmorrhage always takes place within a pocket; the result is that the defence reaction defeats its own object and a condition of ischæmia follows—a condition which favours the growth and invasion of fuso-spirochætal organisms.

Though the organisms, once a breach of the surface has occurred, are able to spread and to undermine the adjacent epithelium, clinical experience shows that acute fulminating ulceration is less common than a persistent subacute type. Whether this is due to the virulence of the organisms or the characteristics of the environment in which they find themselves—the seed or the soil—has not been determined. Stammers states that after the acute infection has subsided, either naturally or because of treatment that has been given, Nature attempts to produce healing in her usual way—by the formation of granulation tissue which is gradually covered by epithelium and progressively fibrosed. But conditions are such that Nature has no chance of success—"one part is healing while another is breaking down, the process of repair is constantly being interrupted, and Nature is constantly trying to catch up with a situation which is never under control".

From the account that has been given of Stammers's work it will be clear that mechanical cleansing and the eradication of pockets are the essentials of treatment. It will also be clear that "the most likely factor predisposing to re-infection is the alteration of structure of the gingival gum caused by healing under adverse conditions". Therapy in this account of Stammers's work must take second place; it is of prime importance to understand the nature of the pathological process.

[1] *The British Dental Journal*, July 5, 1946.

# Abstracts from Medical Literature.

## GYNÆCOLOGY.

### Cervical Hæmorrhage after Subtotal Hysterectomy.

J. E. DAVIS AND D. B. CHEEK (*The Journal of the American Medical Association*, July 6, 1946) have studied 87 cases of cervical stump hæmorrhage occurring between January 1, 1938, and July 1, 1945. Forty of the patients were found to have a cervical cancer; of six of these forty, the cervix appeared normal at the time when they first presented themselves with the complaint of hæmorrhage. The authors point out that hæmorrhage from the cervical stump after subtotal hysterectomy should be investigated. Regardless of a history suggesting the presence of menstruating endometrium, regardless of the appearance of the cervix, regardless of œstrogen therapy, every patient who suffers from such hæmorrhage at any time after the operation should undergo a biopsy of the cervix and gentle curettage of the cervical canal. The authors believe that many subtotal hysterectomies are performed in the presence of an early carcinoma of the cervix, which often proliferates without signs or symptoms. They hope that pre-operative study will decrease the number of cases in which this occurs.

### Vaginal and Cervical Cytology in the Diagnosis of Uterine Cancer.

THE cytological smear of the vagina or cervix permits an expert cytologist to make a reliable presumptive diagnosis of cancer in over 90% of cases. J. E. Ayre (*American Journal of Obstetrics and Gynecology*, June, 1946) found that cancer cells were more readily found in the cervical smear which gave an average error of 6% in 100 cases of known malignant disease. The method is of value in the detection of early cancer of either the cervix or *fundus uteri*, in the assessment of radiation therapy and in the follow-up of cancer patients after treatment. A centrifuge cytology technique for the recovery and staining of cancer cells is described.

### The Artificial Menopause and Cancer of the Breast.

L. HALBERSTAEDTER AND A. HOCHMAN (*The Journal of the American Medical Association*, July 6, 1946) present a study of sixty women suffering from cancer of the breast who were treated by irradiation of the ovaries for the production of an artificial menopause. The authors do not use this form of treatment as a routine measure, but only as a rule when metastases can be shown to have developed. In only a few cases do they perform ovarian sterilization as a preventive measure. The tumours were graded according to Steinthal's classification; six were of grade I, 28 were of grade II and 19 were of grade III; the classification of seven is not given. Thirty-four of the women benefited from the treatment. However, the improvement due to the interruption of œstrogenic secretion from the ovaries is of short duration, since œstrogenic secretion from other sources intervenes. The authors present the results also graded according to the type of growth. Of eighteen patients with adenocarcinoma, the condition of thirteen was improved; of eleven patients with *carcinoma simplex*, that of one was improved; of two patients with scirrhous carcinoma, that of both was improved; one patient had a fibrocarcinoma, and benefited from the treatment; three patients had carcinomata, and two benefited; intracanalicular carcinoma was present in two cases, and improvement occurred in one; there was one case of *carcinoma solidum*, in which improvement occurred; the same result was obtained in the one case of undifferentiated carcinoma; in 21 cases no microscopic examination was made. Thus typical adenocarcinomata are more susceptible than anaplastic cancer of the breast to the œstrogen-inhibiting influence of the artificial menopause. A review of the literature is included.

### Cancer and Prolonged Œstrogen Therapy.

M. FREMONT-SMITH, J. V. MEIGS, R. M. GRAHAM AND H. H. GILBERT (*The Journal of the American Medical Association*, July 6, 1946) report the case of a woman suffering from asthma who was under hospital observation almost continuously for eighteen years. When the patient was aged forty-three years, a connexion between the onset of amenorrhœa on several occasions and increased severity of the asthma was suspected; treatment with œstrogenic substance was begun and continued for almost eight years. Symptoms suggestive of cancer developed, and finally the uterus was removed and found to be the site of an adenocarcinoma (grade II). The authors believe that this is the first report of a case of endometrial cancer occurring in conjunction with a prolonged period of treatment with œstrogen. They point out that their case does not prove, but does strongly suggest, that the unopposed action of œstrogens on the breast and endometrium of certain women may result in the development of cancer. This patient was shown, by the finding of atrophic vaginal epithelium after the œstrogen was withdrawn, to be producing little or no œstrogen, and a strong supposition exists that endometrial cancer might not have developed without the administration of extrinsic œstrogens. Œstrogenic substance at the present time is prescribed for a host of gynæcological complaints, and is not always preceded by a pelvic examination. The physician prescribing œstrogen for women during the menopause should understand the possible danger of prolonged treatment without adequate periods of rest.

### Untoward Effects of Œstrogenic Therapy in the Menopause.

J. S. HENRY (*The Canadian Medical Association Journal*, July, 1945) suggests a more rational therapy of the menopause than is often practised. He maintains that this should be based on an understanding of the changes in ovarian function which bring about the symptoms that require correction, as well as of the relationship existing between the ovary and the anterior lobe of the pituitary. He points out that the ovary has an important function apart from that of providing fertilizable ova during the reproductive period, namely, that of providing for the growth and nutrition of all the reproductive organs. It also regulates the secretion of the gonadotropic hormones of the anterior lobe of the pituitary. The vegetative function of the ovary is carried out by the process of partial follicular development and atresia, which goes on from birth until the end of menstrual function or even later. This supplies the body with gradually increasing amounts of œstrogen before puberty. In some women the supply of follicles is not exhausted at the time of the menopause and this process continues. Thus they are enabled to "grow old gracefully" by a gradual process of diminution of œstrogen. These women have few symptoms at the menopause and rarely require treatment. In others, a sudden withdrawal of œstrogen throws the hormonal balance suddenly out of action and results in various disturbing symptoms. These are the patients who can be helped by supplementary œstrogen therapy. But it is essential that this should follow the natural pattern of cyclical supply and gradual diminution. If this is not done, treatment may do more harm than good, sometimes even prolonging the menopause or leading to the dangers of overstimulation. These latter include uterine hæmorrhage resulting from glandular cystic hyperplasia of the endometrium. They may even lead to malignant changes in any of the reproductive organs. The œstrogens are closely related chemically to some of the carcinogenic substances of coal tar, and in animals they have been proved to play a role in carcinogenesis. Overstimulation by hormones can be brought about not only by excessive dosage, but also by undue prolongation of treatment.

### Testosterone Propionate and Ovarian Carcinoma.

JAMES WYATT (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1945) presents two cases of ovarian carcinoma in which treatment with testosterone propionate was tried. One patient was aged fifty-one years, the other seventy-five years. The first patient, who had had a radium menopause seven years previously, complained of vaginal hæmorrhage. Seventeen years previously she had had a carcinomatous left breast removed. She had had four normal pregnancies. Laparotomy revealed a cyst growing from the right ovary; the other pelvic viscera were normal, and no free fluid or other evidence of malignant disease was present. Investigation of the removed cyst revealed adenocarcinoma. A full course of deep X-ray therapy was given to the pelvis, and three months later the patient was well. Eleven months later again she was readmitted to hospital, on account of recurrence; the pelvis was filled with a mass of growth, and secondary nodules were scattered over all the viscera. Treatment with testosterone propionate was begun and lasted for a little over two months. Injections were given daily at first and then on alternate days, until a total of 6,750 milligrammes had been given. She felt better, gained a little weight and went home, further injections being given to bring the total dosage to 7,200 milligrammes. However, her condition rapidly deteriorated; she suffered from severe epigastric pain and frequently vomited, and she was readmitted to hospital about six weeks after having left it, and died about two months later.

No deepening of the voice or growth of hair on the face or body developed as a result of treatment. The second patient was too ill for major operative procedures, but a small subumbilical incision was made and a large ovarian tumour was found with necrotic walls; a small piece was removed for biopsy. There was no sign of local peritoneal spread. Testosterone propionate was given to a total dosage of 3,150 milligrammes. For the first fortnight she felt better, but then her condition deteriorated and she died about two months later. In this case post-mortem examination revealed that the primary tumour might have been in the large intestine. The author comments that, although treatment with testosteron propionate had the immediate effect of giving the patients a feeling of well-being, this was only temporary. There was no evidence of retrogression in the growth itself or in the metastatic deposits, either clinically or histologically.

### Adenocarcinoma of the Uterus.

L. A. Gray, M. Friedman and W. S. Randall (*Surgery, Gynecology and Obstetrics*, April, 1946) present some observations on a series of ten patients suffering from uterine adenocarcinoma treated over a period of three years. One of the authors designed a special radium applicator, called the hysterostat, to achieve a more equable distribution of radiant energy to all parts of the uterus. In nine of the ten cases the lesions were moderately advanced or advanced, but without induration of the pelvic ligaments; they were therefore considered operable. Carcinoma protruded from the os in two cases, and ovarian metastasis was subsequently found in three cases. On microscopic examination, moderate differentiation (grade II) was found in four cases, moderate undifferentiation (grade III) in four cases, and complete undifferentiation in two cases. In seven cases radium treatment was followed by panhysterectomy and bilateral salpingo-oophorectomy. These patients received unusually large doses of radium; in milligramme hours, the figures were respectively 6,030, 7,000, 7,000, 8,000, 8,900, 10,410 and 12,480. In the superficial myometrium of six of the seven excised uteri nests of apparently viable cancer cells were found; deeper extension was present in three. The one uterus in which no residual carcinoma was found was the site of a highly malignant, undifferentiated adenocarcinoma; the dosage of radium had been 12,480 milligramme hours in two exposures. One patient with early undifferentiated carcinoma was treated by surgical means alone. The authors give no five-year follow-up figures (i) because of the finding of residual carcinoma in six of the seven uteri from patients treated with enormous doses of radium by means of the hysterostat, and (ii) because of the presence of ovarian metastases in three patients, which in their opinion emphasizes the need for oophorectomy as part of treatment. They believe from their study that radium treatment alone will not completely eradicate adenocarcinoma of the uterus, and that subsequent excision of the uterus, Fallopian tubes and ovaries is necessary. They also find that the exact role and specific indications for preoperative radium therapy have not yet been satisfactorily delineated.

## OBSTETRICS.

### Puerperal Infection.

R. Gordon Douglas and Ione F. Davis (*American Journal of Obstetrics and Gynecology*, March, 1946) show that although a great decrease in maternal mortality has occurred from 1935 to 1944 in New York City, the deaths from puerperal septicæmia have remained constant. The process of infection actually commences with the onset of labour in every case, and every effort should be made to combat it by the treatment of ante-partum anæmia and intercurrent disease, by the prevention and restoration of blood loss, by the avoidance of trauma, by the application of surgical principles to labour and delivery, and by the performance of Cæsarean section after not more than eight to twelve hours of labour. Prophylaxis of infection is undoubtedly more important than treatment of established disease, and sulphadiazine and penicillin must be given early to be efficacious.

### An Investigation into Maternal Mortality.

In a review of the maternal mortality at the Chicago Lying-in Hospital for the years 1931 to 1945, M. E. Davis and T. G. Gready (*American Journal of Obstetrics and Gynecology*, April, 1946) find that infection was the principal cause of death, accounting for 32 of the total of 81 maternal deaths; hæmorrhage and shock contributed thirteen and toxæmias of pregnancy six. Heart disease accounted for thirteen of the deaths. Careful analysis indicated that preventible factors were present in two-thirds of the fatal cases. They conclude that infection is rapidly disappearing as a cause of death in obstetrics, and attribute this to the injection of sulphonamides and penicillin and the liberal use of blood, of plasma and of parenterally administered fluids. These combined with proper obstetrics and hospitalization in a well-equipped and well-staffed hospital have resulted in the delivery of almost 20,000 women without a single death due to puerperal infection. An excessive loss of blood influenced directly or indirectly the clinical course of almost half of these women who lost their lives in childbirth, and the unwarranted use of pituitary extract prior to delivery of the baby was the most important cause of serious damage to the reproductive tract. Heart disease shared second place with hæmorrhage as a cause of death, and now must be coupled with infection and toxæmias as a major cause. The management of the obstetric patient with heart disease is the most important factor in the ultimate outcome.

### Bronchopneumonia following Ether Anæsthesia in Obstetrics.

H. C. Hartzell and E. P. Mininger (*Surgery, Gynecology and Obstetrics*, April, 1946) have investigated a series of twenty cases of bronchopneumonia occurring a few days after delivery. The condition was variable in extent, transient, and unassociated with clinical evidence of serious illness. It was nearly always discovered by X-ray examination of the chest made on account of slight fever or of râles in the chest developing after delivery. The anæsthesia was induced by ether usually given by the "open" method and preceded by the administration of varying quantities of barbiturates and other forms of premedication. The chief cause of the bronchopneumonia was the aspiration of gastric contents as a result of vomiting. The authors believe that three other factors may have been involved: (i) pre-anæsthetic medication, favouring suppression of the cough reflex; (ii) prolonged gastric evacuation in labour; (iii) fluidity of the gastric contents, favouring their dissemination in the bronchial tree. The lesions of bronchopneumonia varied in extent from small, isolated patches to extensive bilateral infiltration; they were found in all parts of the lung fields. They caused no serious illness, and no fatalities resulted. Sulphonamide treatment seemed ineffective. The radiological problem was one of diagnosis, the condition requiring differentiation from pulmonary tuberculosis, sarcoidosis, other forms of pneumonia, pulmonary œdema and atelectasis. With reference to prevention and treatment, the authors state that the measures of postural drainage of the bronchial tree and its aspiration by catheter suction or bronchoscopically are the recognized methods of treating vomiting and aspiration during anæsthesia. The administration of an emetic in early labour to a patient who has recently eaten solid food has been practised, in the hope that such gastric evacuation may prevent post-anæsthetic vomiting and aspiration of particulate matter. Generally the properly trained anæsthetist will maintain his patient in the narrow zone of anæsthesia in which labour processes can continue without the development of vomiting.

### The Hogben Pregnancy Test.

E. C. Foote and G. E. Seegar Jones (*American Journal of Obstetrics and Gynecology*, May, 1946) have used the Hogben diagnostic test of pregnancy, based upon egg extrusion in the South African clawed toad, in 157 clinical cases. Ovulation occurs six to eighteen hours after the injection of a specimen containing concentrated chorionic gonadotropin. The authors' results showed that before the fortieth day of gestation, the test was inaccurate, but thereafter the accuracy was 96%. In two-thirds of the cases of threatened and incomplete miscarriages a positive result was obtained; *corpus luteum* cysts gave a negative result. The advantage over the Friedman test is that it gives no false positive results.

### Causes of Breech Presentation.

Pendleton Tomkins (*American Journal of Obstetrics and Gynecology*, May, 1946) ascribes breech presentations to extension of the legs not allowing free movement of the fœtus. Breech babies are often babies who have not moved actively during pregnancy and particularly in the latter months. The intrauterine vigour of the fœtus may be impaired as a result of hydrocephalus, meningocele, dysplasia of the hips, cerebral agenesis and other congenital defects; in many of these conditions breech presentation is accompanied by polyhydramnios. The author shows that the "classical factors" (contracted pelvis, pelvic tumours, *placenta prævia* and fœtal malformations) account for only one-sixth of the breech births among mature single infants.

# Bibliography of Scientific and Industrial Reports.[1]

## THE RESULTS OF WAR-TIME RESEARCH.

*During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken with former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.*

*Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) Those marked with an asterisk may be obtained by approved applicants without cost on making application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.*

*Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.*

---

Documents and equipment obtained by investigation of German X-ray and associated industries. Siemens' "Metal-Searcher" (for locating metal foreign bodies); technical articles regarding equipment and techniques; technical write-up and instruction manual. Wiring diagram and assembly and cross section drawings of probes. Off. Pub. Bd., Report, PB 2177. No date. 39 pp. Price: Microfilm, 50c.; Photostat, $3.00.

The articles included in this report are entitled: 1. *Ein Hochfrequenzgerät zum Aufsuchen metallischer Fremdkörper*, by Dr. J. Pätzold (reprint from *"Der Chirurg"*, 13, *Jahrgang*, 1941, *Heft* 7); 5 pages. 2. *Ein Hochfrequenz-verfahren zum Auffinden von Metallfremdkörpern*, by Doz. Dr. G. Küntscher and Dr. A. Jaumann (*Sonderabdruck aus Nr.*, 50, 1940); 7 pages. 3. *Gebrauchs-Anleitung Siemens-Metallsucher*, no date; 8 pages. 4. *Was musz der Chirurg beim Gebrauch des Siemens-Metallsuchers Beachten?*, no date; 15 pages.

Documents and equipment obtained during investigation of German X-ray and associated industries. Siemens' *"Ultraschall-generator"* (supersonic frequency unit for treatment of certain nerve conditions); description of equipment and instruction manual (and translation), photographs of unit, and assembly and cross section drawing of Piezo-electricsupersonic generator. Off. Pub. Bd., Report, PB 2179. No date. 13 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report is a detailed instructions manual for the mounting and operation of the Supersonic Wave-Generator (*Ultraschallgenerator*). Thanks to the simple mode of operation and the fact that a hooded quartz is used, the apparatus is especially suitable for electric therapy, notably for use on the human body.

McCarthy, Charles L. Venereal disease control office for Thuringia. Off. Pub. Bd., Report, PB 1785. 1945. 23 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This report gives a description of the Office for the Control of Venereal Diseases at Weimar in the State of Thuringia. The office is well directed and functions in a satisfactory manner. The standard of treatment, the requirements for cure, the issuance of marriage certificates and the follow-up of the so-called cured cases are satisfactory. The control of refractory patients, although not supported by law, is quite good. Some doubt exists about the reliability of using Chediak slide tests to diagnose syphilis even though positive or doubtful findings are controlled by Wassermann tests. It is possible that some cases are missed by the Chediak test. Syphilis occurs in 0·01% of all German civilians and in 1·30% of displaced persons of all nationalities in the State of Thuringia. There has been a 35% increase in syphilis in Germans from 1943 to 1944; however, the comparison of 1944 with 1940 shows only a 25% increase. Gonorrhœa shows a 28% increase from 1940 to 1943. Between 18% and 20% of the total number of cases of gonorrhœa and of syphilis are never reported and account for many relapses and the spread of infection.

Simons, H. The importance of the Thedan-Blue method for tropical medicine, together with instructions for its use. Off. Pub. Bd., Report, PB 2045. 1945. 17 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This is a report in German on the Thedan-Blue staining method. The Thedan-Blue stains are applicable to certain blood portista, especially trypanosomes, spirochætes (recurrens type), leptospiral organisms, and certain forms of malaria plasmodia. It is reported to be a purely diagnostic method for rapid, reliable and cheap mass examination. It is stated that all the parasites mentioned can be diagnosed without the use of oil immersion, and that the staining of cover-glass preparations, and of thick drops, may be carried out in less than one minute. The report is illustrated by twelve pictures showing various preparations stained by this method. For details the original report should be consulted.

United States Surgeon-General's Office. Medical documents, reprints and books on microfilm (materials listed are available on microfilm, PB 1686-PB 1703). Off. Pub. Bd., Report, PB 1704. 1945. 41 pp. Price: Microfilm, 50c.; Photostat, $3.00.

List of medical materials collected by American investigators and available on microfilm (PB 1686-PB 1703). These documents, some of which are unpublished, cover a wide variety of medical subjects. Microfilms (22 reels) are in German.

Volwiller, Ernest H. Investigations by Professor Kuhn, *Kaiser Wilhelm Institut für medizinische Forschung*. Off. Pub. Bd., Report, PB 1789. 1945. 15 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This is a supplementary report on Professor Kuhn's investigation on biological and medical problems. The report deals with cyclodecapentæne, vitamin research, dibromosalicyl, sulphones, sulphonamide antagonists, local anæsthetics, and proteolytic enzymes. Attached to the report as appendices are reports of investigations on the nature and action of alleged samples of cyclodecapentæne carried out in American laboratories. Vitamin *A* has not been studied recently by Kuhn, except in relation to the supply and stabilization of carotenes. During the last few years vitamin *E* has had considerable attention in Kuhn's laboratory. In doses much larger than normal, it was found to prevent and to cure fatty degeneration of the liver in rats fed on an *E*-deficient diet. Vitamin $B_4$ has also received attention. Citrin has not been studied, but rutin, obtained from tobacco, has been investigated. Much work has also been done on vitamins as components of culture media for bacteria. Dr. Otto Dann in Kuhn's laboratory has made compounds similar to *p*-aminobenzoic acid. These are more unstable than PAB, however, and do not exhibit an antagonistic action as does PAB. Kuhn's laboratory has also made the thiophene analogue of procaine, but it is not a very good local anæsthetic.

Kaiser Wilhelm Institut fur Arbeitsphysiologie. Reprints dealing with industrial physiology and industrial hygiene. Off. Pub. Bd., Report, PB 1706. 1942-1944. 797 pp. Price: Microfilm, $8.00; Enlargement Print, $54.00.

This report consists of microfilmed articles from the following publications.

*Arbeitsphysiologie* 11 (1941): *Zur Frage der Arbeits-und Pausengestaltung bei Fliessarbeit*, by Otto Graf, pp. 503-519 (21 pp.).

*Arbeitsphysiologie* 12 (1942-1943): *Arbeitsphysiologische Prüfung beim Mähen mit einer neuen Sense*, by Alexander Szakall, pp. 1-14 (18 pp.). *Zur Frage der Arbeits-und Pausengestaltung bei Fliessarbeit*, by Otto Graf, pp. 19-30 (14 pp.) and pp. 332-376 (14 pp.). *Adrenalin und Arbeit*, by Gunther Lehmann and Hans Ferdinand Michaelis, pp. 52-80 (34 pp.), pp. 264-271 (9 pp.), pp. 298-312 (15 pp.) and pp. 440-448 (9 pp.). *Der Einfluss der Tourenzahl beim Radfahren auf die maximale Sauerstoffaufnahme und auf die Ausnutzung des Atemsauerstoffs*, by Hedwig Michaelis and E. A. Müller, pp. 81-84 (4 pp.). *Die Bedeutung des alveolaren* $CO_2$*— Druckes für die Bestimmung des auf die Atmung entfallenden Energieverbrauches*, by Hedwig Michaelis and E. A. Müller, pp. 87-91 (11 pp.). *Die Pulszahl als Kennzeichen für Stoffaustausch und Erdmudbarkeit des Arbeitenden Muskels*, by E. A. Müller, pp. 92-104 (17 pp.). *Eine verbesserter Spiro-*

---

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

*graph*, by E. A. Müller and Alfred Müller, pp. 120-123 (4 pp.). *Die Wirkung von Traubenzucker und Traubenzucker $B_1$-Kombinationen auf die Leistungsfähigkeit bei Hitzearbeit*, by Werner Droese, pp. 124-133 (11 pp.). *Über die Wirkung einer warmen Mahlzeit auf die Leistungsfähigkeit von Frauen bei Nachtarbeit*, by Hans Ferdinand Michaelis, pp. 134-141 (8 pp.). *Untersuchungen über den Grad der geistigen Beanspruchung* I-II, by E. Bornemann, pp. 142-191 (48 pp.), pp. 142-343 missing. *Der Energieaufwand für die Atmung beim Menschen*, by E. A. Müller, Hedwig Michaelis and Alfred Müller, pp. 192-196 (5 pp.). *Der Calorienbedarf der Berufe, ermittelt aus den Erhebung von Wirtschaftsrechnungen im Deutschen Reich vom Jahre*, 1927-28, by Heinrich Kraut and Herbert Bramsel, pp. 197-221 (25 pp.). *Der Eiweissverbrauch des deutschen Volkes im Jahre*, 1927-28, *berechnet auf Grund der Erhebungen von Wirtschaftsrechnungen in* 2,000 *Haushaltungen*, by Heinrich Kraut and Herbert Bramsel, pp. 222-237 (16 pp.). *Der Kohlehydrat- und der Fettverbrauch des deutschen Volkes im Jahre*, 1927-29, *berechnet auf Grund der Erhebungen von Wirtschaftsrechnungen in* 2,000 *Haushaltungen*, by Heinrich Kraut and Herbert Bramsel, pp. 238-248 (11 pp.). *Eine arbeitsphysiologie Untersuchung der Melkarbeit*, by Hedwig Michaelis and E. A. Müller, pp. 249-263. *Die Abhangigkeit des Energieverbrauchs von der alviolaren $CO_2$-Spannung bei Ruhe und Arbeit*, by E. A. Müller, Hedwig Michaelis and Alfred Müller, pp. 313-319 (7 pp.). *Der Pulsquotient als Mass der Muskelermüdung*, by E. A. Müller, pp. 320-331 (13 pp.). *Eine Methode zur Untersuchung der pharmakologischen Beeinflussung von Koordinationsleistung*, by Otto Graf, pp. 449-469 (21 pp.).

*Arbeitsphysiologie* 13 (1943-1944): *Arbeitsphysiologische Untersuchungen am Schubkarren*, by E. A. Müller and Alfred Müller, pp. 1-8 (8 pp.). *Maximale Leistung und Maximale Arbeit*, by Alexander Szakall, pp. 9-41 (33 pp.). *Die Wirkung von Insulin und Muskelarbeit aus das Magnesium in Blutserum und Harn*, by Norbert Hamar and Alexander Szakall, pp. 42-48 (7 pp.). *Die Veränderungen der obersten Hautschichten durch den Dauergebrauch einiger Handwaschmittel*, by Alexander Szakall, pp. 49-56 (8 pp.). *Adrenalin und Arbeit*, by N. Hamar, H. F. Michaelis and A. Szakall, pp. 57-62 (6 pp.). *Experimentalle Untersuchung über die Vitamin $B_1$-Versorgung der grosstädtischen Bevölkerung in den Jahren*, 1941-42 *und* 43, by Werner Droese, pp. 63-78 (16 pp.). *Zur Frage der Monotonie der Arbeit und ihrer Bekämpfung*, by Otto Graf, pp. 95-100 (6 pp.). *Versuche über die Hebung der Leistungsfähigkeit durch Ultraviolettbestrahlung*, by Gunther Lehmann and Alexander Szakall, pp. 101-113 (13 pp.). *Über des verhalten des Harn pH bei langdauernder Muskelarbeit und anschliessende Erholung*, by Nobert Hamar and Alexander Szakall, pp. 114-124 (12 pp.). *Ein Verfahren zur Zwanglosen Steuerung der Arbeitsgeschwindigkeit nach vorgegebenen Arbeitskurven insbesondere zu physiologisch richtiger Arbeitsablaufreglung*, by Otto Graf, pp. 125-139 (15 pp.). *Über das Verhalten des Blut-und Harnkaliums während Muskelarbeit und der darauffolgenden Erholung bei untrainierten und trainierten Hunden*, by Norbert Hamer and Alexander Szakall, pp. 140-150 (12 pp.). *Kalium-Wasser-und Kreatininaussonderung untrainierter Hunde während Muskelarbeit und in der Erholung*, by Norman Hamar, pp. 151-156 (6 pp.).

*Arkiv f. experim. Pathologie und Pharmakologie*, 201-202 (1943): *Einfluss der Narkose auf den Adrenalingehalt des Blutes*, by Hans Ferdinand Michaelis, 201: 580-588 (10 pp.). *Tabakgenus und Adrenalinspiegel*, by Gunther Lehmann and Hans Ferdinand Michaelis, 202: 627-632 (6 pp.).

*Biochemische Zeitschrift* 312 (1942): *Zum Kohlenhydratstoffwechsel der $B_1$-Avitaminotischen Taube*, by H. Kraut and M. Rohdewald, pp. 289-307 (19 pp.).

*Biochemische Zeitschrift* 315 (1943): *Kalkbilanz und Kalkbedarf*, by H. Kraut and H. Wecker, pp. 329-344 (16 pp.).

*Biochemische Zeitschrift* 316-317 (1943-1944): *Über die Verträglichkeit von synthetischen Fett aus Fettsauren mit 6-bis 12 C-Atomen*, by H. Kraut, A. Weischer and R. Hügel, 316, pp. 96-107 (1943), (13 pp.); 317, pp. 187-192 (1944), (6 pp.). *Über den Bleigehalt der Haare*, by Heinrich Kraut and Marta Weber, 317, pp. 133-148 (1944), (16 pp.).

*Der Bergbau vereinigt mit Kohle und Erz*, July 2, 1942: *Untersuchungen über die Zusammeenhänge zwischen Arbeitsführung und Leistungsteigerung*, by H. Vennewald, pp. 1-8.

*Forschungen und Fortschritte* 18 (1942): *Kristallbau und Leitvermogen der Metalle*, by Eduard Justi, pp. 65-70 (6 pp.). *Die Ursachen der Erhaltung bronzezeitlichen bekleideter Leichen in Baumsärgen*, by Karl Gripp, pp. 70-72 (3 pp.). *Das Zeusbild des Phidias in Olympia*, by Hans Schräder, pp. 72-73 (2 pp.). *Die Enstchung der italienischen Stadtkommunen des Mittelalters*, by Walter Goetz, pp. 73-75 (3 pp.). *Korperform und Sozialer Aufstieg*, by Ilse Schwidetzky, pp. 75-76 (2 pp.). *Über die absoluten Mengen des beim Rauchen aufgenommenen Nikotins*, by Adolf Wenusch, pp. 77-78 (2 pp.). *Neueres zum Feldspatproblem*, by Herman Tertsch, pp. 78-79 (2 pp.). *Spiralen im Eis*, by Friedrich Trey, pp. 79-80 (2 pp.). *Menschliche Leistung und Leistungsfähigkeit*, by Gunther Lehmann, pp. 80-82 (3 pp.). Obituaries of Hans Lippe, Walther Nernst and Friedrich von Müller, pp. 82-84 (3 pp.).

*Hoppe-Seyler's Zeitschrift für physiologische Chemie* 275 (1942): *Die Bestimmung der Kieselsäure im Blut*, by Heinrich Kraut and Marta Weber, pp. 127-134 (12 pp.).

*Luftfahrtmedizin* 9 (1944): *Ultravioletbestrahlung und Höhenfestigkeit*, by G. Lehmann and E. A. Müller, pp. 37-43 (7 pp.). *Über die Ursachen der individuellen Unterschiede der Höhenfestigkeit*, by E. A. Müller and Hedwig Michaelis, pp. 44-48 (5 pp.).

*Pflügers Archiv für die gesamte Physiologie des Menschen und der Tiere* 248 (1944): *Die Einfluss lokaler Kältereize auf den Stoffwechsel*, by E. A. Müller, pp. 521-526 (6 pp.).

*Stahl und Eisen* 64 (1944): *Die Bewertung der körperlichen Arbeit aus physiologischer Grundlage*, by Gunther Lehmann, pp. 85-90 (6 pp.).

*Deutsche medizinische Wochenschrift* 49 (1941): *Probleme der Ermudungsbekampfung*, by E. A. Müller, pp. 1340-1347 (8 pp.).

*Zeitschrift die Ernahrung Beihefte* 8 (1943): *Vitamintabellen*, by Werner Droese and Herbert Bramsel, Ed. 2, 93 pp.

*Zeitschrift für Kreislaufforschung* 34 (1942): *Herzschwäche und Herztonus*, by E. A. Müller, pp. 297-304 (8 pp.).

EUSTIS, WARNER. Manufacture of adhesive surgical dressings at Sander Chemical and Pharmaceutical Ges. M.B.H., Munich. Off. Pub. Bd., Report, PB 2457. 1945. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report deals with the ownership, operations, processes and products of Sander firm, manufacturer of small adhesive surgical dressings of good quality. Principal chemicals used for adhesives are: Plexigum, Vinnapas, Essigester Solven, Oppanol, Palatinol, "Tributylphosphate", Colophony. Sander dressings cannot be heat sterilized because of heat sensitivity. However, picric acid impregnation is used on all gauze, it is believed, where such gauze touches a wound.

EUSTIS, WARNER. Manufacture of sanitary napkins and allied products at Camelia works, Vereingte Papierwerke Nurnberg, Nurnberg. Off. Pub. Bd., Report, PB 2455. 1945. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report describes ownership, management, foreign patents, foreign agreements, manufacturing processes and products of this company, reputed to be the largest European manufacturer of sanitary napkins, cellulose wadding, toilet paper, paper handkerchiefs, box board and corrugated box board. Foreign patents owned are listed.

EUSTIS, WARNER. Manufacture of surgical dressings and allied products at Paul Hartmann Co., Heidenheim. Off. Pub. Bd., Report, PB 2460. 1945. 19 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This report describes history, ownership, management and operations of the largest and best manufacturer of surgical dressings in Germany. Foreign sales agents and representatives, principal foreign customers, foreign patents and production for foreign consumption and export are outlined. Equipment and processes in bleaching, drying, carding, dressing, knitting, packaging, and adhesive departments are described. In addition to its own products, this concern carried, as a jobber, additional items. Photographs and catalogues referred to in the report have been forwarded to Lieutenant-Colonel Stephen J. Kennedy, Office of Quartermaster-General, Military Planning Division, Washington, D.C. For another report on this subject see PB 2448, page 274.

EUSTIS, WARNER. Manufacture of sutures and ligatures at Dr. Hammer Co., Hamburg. Off. Pub. Bd., Report, PB 2452. 1945. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report presents information on ownership, management, products, manufacturing methods and packaging. Specifications of products are given and types of bottles used for packaging are described. Products are useful, but their quality has not been definitely established.

EUSTIS, WARNER. Manufacture of surgical adhesive and dressings and artificial limbs by Julius Teufel Company, Stuttgart. Off. Pub. Bd., Report, PB 2459. 1945. 8 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report contains information on ownership, management, location of factories, patents, domestic and foreign sales, and products. The company has several plants on the outskirts of Stuttgart engaged in the manufacture of artificial limbs, surgical dressings, stockings for the stumps of limbs, elastic sanitary garments, and adhesives of the heat-sensitive type. The formula for special heat-sensitive adhesive made by Teufel is given.

# British Medical Association News.

## NOTICE.

THE General Secretary of the Federal Council of the British Medical Association has announced that the following medical practitioner has been released from full-time duty with His Majesty's Forces and has resumed civil practice as from the date mentioned:

Dr. R. F. A. Becke, "Locarno", 141, Macquarie Street, Sydney (September 23, 1946).

# Medical Societies.

## THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the Institute of Medical and Veterinary Science, Adelaide, on June 14, 1946.

### Some Aspects of Paramyotonia Congenita.

DR. J. S. COVERNTON AND DR. M. H. DRAPER presented a paper on some aspects of *paramyotonia congenita*. They pointed out that myotonia was a rare anomaly of muscular function in which difficulty in relaxation followed voluntary activation. This difficulty was usually improved with repetition of movement, and was made worse by cold, by emotional excitement, and in female subjects by menstruation and pregnancy. This disorder was hereditary and was transmitted as a Mendelian dominant inheritance.

The cases exhibiting this muscular disorder fell into three categories, all of which were probably the result of one underlying defect. They were: (i) *Myotonia atrophica* or *dystrophica myotonica*, exhibiting myotonia together with muscular atrophy of a peculiar distribution. (ii) *Myotonia congenita* or Thomsen's disease, exhibiting mytonia with muscular hypertrophy. (iii) *Paramyotonia congenita* (*myotonia intermittens*) in which the myotonic symptom was modified in that increasing functional capacity was not obtained in strong repeated movement as occurred in (i) and (ii). On the contrary strong-willed movement produced a progressive difficulty in relaxation which ultimately resulted in complete powerlessness of the limb undergoing movement.

The influence of various drugs on the myogram of a paramyotonic subject revealed that adrenaline and "Prostigmin" worsened the condition. Strychnine and potassium citrate had a slight worsening effect. "Moryl" had first an improving and then a worsening effect. "Syntropan" reversed the worsening of "Prostigmin" and generally improved the condition. Alcohol, hyoscine, benzedrine, phenobarbital, atropine sulphate, ergotamine tartrate and strangely quinine (which relieved *myotonia congenita*) had no significant effect.

Electromyographic studies in which a double beam cathode ray oscilloscope was used supported the contention that the fundamental defect lay in the muscle fibre.

It had recently been proven that myotonia existed in a form similar to Thomsen's disease in a strain of goats.

Stringhalt was a common hereditary disease in horses, and its manifestations were associated with spasms in voluntary muscles. These spasms were worsened by cold and fright and did not improve markedly with repetition. Stringhalt was worsened by "Prostigmin" and quinine (investigation of one case). The latter raised a possibility of quinine sensitivity. "Syntropan" improved stringhalt. The evidence suggests that stringhalt could be allied to paramyotonia.

In conclusion, it was suggested that as in Thomsen's disease the fundamental defect in paramyotonia lay in the muscle fibre and its ability to respond repetitively to stimuli. In paramyotonia, however, this repetitive response was more easily induced.

The work was done under a grant from the National Health and Medical Research Council.

A meeting of the Medical Sciences Club of South Australia was held at the Institute of Medical and Veterinary Science, Adelaide, on August 2, 1946.

### Ammoniacal Smell in the Stomach Post Mortem in Uræmia.

PROFESSOR J. B. CLELAND read a paper on the occurrence of ammonia in the stomach *post mortem*. He said that whilst making a post-mortem examination in an obscure case in a woman of thirty-three during the Easter holidays, he noticed a strong smell of ammonia, which was thought at first to be a leakage from the refrigeration machinery. The stomach had just been opened by the assistant, and Professor Cleland was about two feet away from it when the smell was noticed, and it was soon traced to the stomach itself. The day being a holiday, the Biochemistry Department was closed and there was no means of ascertaining the amount of ammonia. However, red litmus was turned deeply blue, and the smell seemed nearly as strong as that of smelling salts, and as had been noted, was picked up at a distance of about two feet.

The patient had died rather suddenly at 6.30 the night before and the post-mortem examination was being made at about 9.30 a.m. next day in the middle of April, the weather being cool. The patient's blood urea nitrogen content had been increasing rapidly during the latter part of her illness, and shortly before death reached 200 milligrammes per 100 millilitres of blood. The patient showed extensive purpuric patches over the body; and hæmorrhages were present in the stomach wall with a considerable quantity of blood in the contents and diffuse hæmorrhages in the rectum. Both lungs were extensively mottled and hæmorrhagic splashes showed in the pelves of the kidneys.

According to the history the patient had been pregnant, and late in the night of April 3, after having attended an ante-natal clinic, she had been awakened by pain in the stomach which gradually had got worse. She began losing blood *per vaginam* and vomited. Her systolic blood pressure was 190 and her diastolic pressure 110 millimetres of mercury and there was much albumin in the urine. She was approximately six months pregnant and was considered to be suffering from some toxæmia of pregnancy. The fœtus and placenta were expelled on April 5. After this she passed only two or three ounces of urine a day, and her blood urea nitrogen content kept on increasing until she finally became extremely drowsy and died rather suddenly. In addition to the hæmorrhages already mentioned, the post-mortem examination showed the uterus to be the size of a coconut with hæmorrhages and necrotic material; much œdema of the hands and feet was present; and the kidneys were enlarged, weighing eleven and a half and twelve ounces, but microscopically they showed cloudy swelling, and some of the tufts were rather shrunken looking.

The ammonia in the stomach had probably been formed after death as a result of the presence in the stomach contents of organisms capable of converting the urea into ammonia.

From time to time in cases of old cystitis in post-mortem examinations that had been conducted not many hours after death, Professor Cleland had noticed that the smell of ammonia had been so strong as to be almost unbearable, but he had not previously come across any noticeable smell of ammonia in stomach contents. He would certainly look for it in future cases where there had been a high blood urea content.

### Torula in the Cerebro-Spinal Fluid.

MR. E. L. FRENCH demonstrated, by means of the microscope and photomicrographs, the Indian ink technique for detecting yeast cells in spinal fluid. The cerebro-spinal fluid was obtained *post mortem* from a patient thought to have had lymphocytic choriomeningitis. Reference was made to a case seen *ante mortem* two years previously in the army, in which penicillin, sulphonamide drugs, flavines and thermotherapy were found to be of no avail.

PROFESSOR J. B. CLELAND briefly outlined the post-mortem findings in the present case. The interesting feature was a lung lesion from which the yeast was later cultivated, and which was considered to be the primary lesion. Professor Cleland drew attention to a previous case in which the primary lesion was also in the lung close to a bullet lodged there many years before owing to an accident.

# Naval, Military and Air Force.

## APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 178, of September 19, 1946.

AUSTRALIAN MILITARY FORCES.

## Australian Army Medical Corps.

NX183 Lieutenant-Colonel (Temporary Colonel) C. R. Blomfield relinquishes the appointment of Assistant Director-General of Medical Services, "A" Branch, Army Headquarters, and is appointed Deputy Director-General of Medical Services, "A" Branch, Army Headquarters, 7th June, 1946.

The following officers are transferred to the Reserve of Officers (Australian Army Medical Corps): Lieutenant-Colonels NX130975 H. M. Owen, 25th May, 1946, and NX109 R. F. A. Becke, 30th May, 1946.

The notification respecting Honorary Captains NX502740 D. K. Lehmann and NX502739 C. Radeski which appeared in Executive Minute No. 116 of 1946, promulgated in *Commonwealth Gazette* No. 100 of 1946, is withdrawn.

*2nd/9th Australian General Hospital.*—The notification respecting SX26444 Major G. H. Solomon which appeared in Executive Minute No. 139 of 1946, promulgated in *Commonwealth Gazette* No. 117 of 1946, is withdrawn.

*22nd Australian Camp Hospital.*—The name of QX60942 Captain (Temporary Major) P. J. Monahan is as now shown and not as it appeared in Executive Minute No. 118 of 1946, promulgated in *Commonwealth Gazette* No. 100 of 1946.

*2nd/2nd Australian Casualty Clearing Station.*—The date of transfer to the Reserve of Officers of VX57663 Major J. H. Colebatch which appeared in Executive Minute No. 130 of 1946, promulgated in *Commonwealth Gazette* No. 117 of 1946, is amended to read "12th April, 1946".

*Interservice Medical Wing Demobilization Centres (Australian Military Forces Component).*—The notification respecting Honorary Captains SX34531 R. Hunter, SX34532 M. K. Smith and SX34533 R. S. Colton, which appeared in Executive Minute No. 118 of 1946, promulgated in *Commonwealth Gazette* No. 100 of 1946, is withdrawn.

VX14845 Lieutenant-Colonel N. M. Eadie relinquishes command of 101st Australian General Hospital (Australian Imperial Force) and is placed upon the Regimental Supernumerary List, 10th May, 1946.

VX133459 Major (Temporary Lieutenant-Colonel) H. E. Pearce is placed upon the Regimental Supernumerary List, 7th June, 1946.

*To be Temporary Major, 7th June,* 1946.—WX33485 Captain W. F. Tomlinson.

*Reserve of Officers.*

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and on the date prior to such transfer, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers:

VX14654 Major L. G. Morton, 23rd May, 1946, and QX55284 Captain J. N. Ure, 16th May, 1946.

*101st Australian General Hospital (Australian Imperial Force).*—Captains VX133260 W. R. Rigg, 22nd May, 1946, and NX146396 G. H. H. Gall, 18th May, 1946.

*No.* 102 (*Holland Park*) *Military Hospital.*—QX35063 Major A. J. Foote, 21st May, 1946, and NX123748 Captain D. B. Wightman, 28th May, 1946.

*No.* 110 (*Perth*) *Military Hospital.*—Captains WX34757 T. Morris, 23rd May, 1946, and WX37474 M. de C. Clarke, 30th May, 1946.

*No.* 112 (*Brisbane*) *Military Hospital.*—QX36368 Captain G. K. Hawkins, 17th May, 1946.

*No.* 113 (*Concord*) *Military Hospital.*—NX137120 Major G. McL. Blaxland, 17th May, 1946.

*No.* 115 (*Heidelberg*) *Military Hospital.*—VX310 Major E. F. Mackenzie, 29th May, 1946, Captains VX148681 D. J. W. Edwards and VX68700 L. O. Morgan, 1st June, 1946.

*118th Australian General Hospital (Australian Imperial Force).*—SX26973 Major N. T. M. Wigg, 28th May, 1946.

*2nd Australian Camp Hospital.*—QX38605 Captain K. J. Shellshear, 24th May, 1946.

*11th Australian Camp Hospital.*—N279596 Captain E. H. V. Lloyd-Williams, 25th May, 1946.

*16th Australian Camp Hospital.*—NF135667 Captain T. L. Latimer, 22nd May, 1946.

*32nd Australian Camp Hospital.*—V147213 Captain F. J. Bryan, 24th May, 1946.

*71st Australian Camp Hospital.*—Captains SX15351 J. C. Yeatman, 30th May, 1946, and WX37237 F. H. Ebell, 23rd May, 1946.

*102nd Australian Casualty Clearing Station (Australian Imperial Force).*—VX102734 Captain W. S. Benwell, 25th May, 1946.

*2nd/8th Australian Field Ambulance.*—NX116628 Captain S. Lackey, 28th May, 1946.

*Interservice Medical Wing Demobilization Centres (Australian Military Forces Component).*—Captains NX201128 J. M. Banks, 18th May, 1946, NX201582 J. C. Andrews, 25th May, 1946, VX93239 J. W. Kent, 30th May, 1946, VX149657 J. W. Cook, 23rd May, 1946, and QX48884 L. L. Grimmett, 16th May, 1946.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers and are granted from such date honorary rank on the Reserve of Officers equivalent to the temporary rank relinquished:

*2nd/11th Australian General Hospital.*—NX14065 Captain (Temporary Major) M. Sterling-Levis, 18th May, 1946.

*No.* 115 (*Heidelberg*) *Military Hospital.*—NX114069 Captain (Temporary Major) G. K. Vincent, 28th May, 1946.

*5th Australian Camp Hospital.*—WX29300 Captain (Temporary Major) A. Siglin, 25th May, 1946.

*102nd Australian Casualty Clearing Station (Australian Imperial Force).*—NX140228 Captain (Temporary Major) C. R. M. Laverty, 23rd May, 1946.

*4th Australian Field Ambulance.*—VX64372 Captain (Temporary Major) A. J. Barnett, 25th May, 1946.

*Retired List.*

The undermentioned officers are placed upon the Retired List on the dates indicated and with permission to wear the prescribed uniform, and, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of placement upon the Retired List:

*No.* 102 (*Holland Park*) *Military Hospital.*—NX171725 Captain W. S. Hemphill, 21st May, 1946.

*70th Australian Camp Hospital.*—NX137660 Captain R. K. Constable, 21st May, 1946.

*Interservice Medical Wing Demobilization Centres (Australian Military Forces Component).*—NX113200 Captain A. E. Diethelm, 21st May, 1946.

*Permanent Supernumerary List.*

The notification respecting the placement upon the Retired List of TX2199 Major E. A. Rogers which appeared in Executive Minute No. 139 of 1946, promulgated in *Commonwealth Gazette* No. 117 of 1946, is amended to read under the above heading.—(Ex. Min. No. 167—Approved 31st July, 1946.)

*Reserve Citizen Military Forces.*

*Queensland Lines of Communication Area.*—The following officers are placed upon the Retired List, 2nd May, 1946: Colonel A. H. Marks, C.B.E., D.S.O., V.D., Lieutenant-Colonels R. S. McGregor, D.S.O., V.D., A. Horn and F. C. Wooster, D.S.O., V.D., honorary Lieutenant-Colonel C. J. Weedon, V.D., Majors J. V. J. Duhig, H. S. McLelland and P. A. Stevens, and Captain R. C. Rutter.

The following officers are retired, 2nd May, 1946: Honorary Captain J. A. Spalding, Lieutenant-Colonel E. D. Ahern, Major J. G. Avery, honorary Captains B. B. Barrack, F. C. Bechtel, J. Bogle and J. Bostock, Captain L. P. Brent, honorary Captain R. O. Bridgeman, honorary Major R. G. Brown, Captain W. G. Brown, honorary Captains E. Bruce-Smith and H. F. C. Brunnich, Captain A. R. Burke-Gaffney, Majors G. F. Cherry, A. S. Clowes and H. Crawford, Captains J. L. Dart, A. Davison, C. B. Deane-Butcher and H. Dolman, honorary Major G. A. C. Douglas, O.B.E., Captains W. J. F. Douglas and D. A. Dowling, honorary Captain R. Evans, Lieutenant-Colonel W. A. Fraser, D.S.O., honorary Captains L. W. Gall, R. Graff and H. W. Hackett, Captain R. J. Haynes, honorary Captains W. J. Hile, H. W. Horn and A. F. Kelly, Captain R. H. Leeds, Major J. Macdonald, O.B.E., Captain G. W. Mason, honorary Captain F. G. Meade, Captain E. Meikle, honorary Captains G. Middleton, P. W. Mitchell and G. H. McCafferty, honorary Major L. M. McKillop, honorary Captains L. J. J. Nye and W. B. O'Connor, Captains S. V. O'Regan, M.C., and E. J. O'Sullivan, honorary Captains J. E. Overstead and V. S. Parer, Captain A. N. Park, Lieutenant F. H. Phillips, O.B.E., Captains F. G. Power and A. J. Reye, honorary Captains A. S. Roe, J. C. Ross and W. J. Saxton, Major J. S. Smyth, honorary Captains A. W. Stark, A. W. St. Ledger and J. C. Thompson, honorary Major E. G. Thomson, Captain C. E. Tucker and honorary Captains T. I. Wallace and H. S. Waters.

XUM

*4th Military District.*—Lieutenant-Colonel E. A. H. Russell, V.D., is placed upon the Retired List, 4th June, 1946.

The following officers are retired, 4th June, 1946: Honorary Captains P. T. S. Cherry, D. R. W. Cowan, E. J. Frayne and J. E. Everard, Lieutenant-Colonel H. K. Fry, D.S.O., honorary Major H. M. Jay, honorary Captains C. E. King and G. C. H. Nicol, Major D. M. Steele, M.C., Captain R. J. Verco.

### Royal Australian Air Force.

*Citizen Air Force: Medical Branch.*

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Squadron Leader T. W. Capell (253179), 13th June, 1946, Temporary Flight Lieutenant P. A. Hamilton (257647), 12th June, 1946, (Temporary Wing Commanders) M. L. Creightmore (291210), 14th June, 1946, V. E. Knight (251197), 21st June, 1946, (Temporary Squadron Leaders) M. H. M. Ryan (252894), 18th June, 1946, H. E. W. Lyons (282726), 19th June, 1946, (Temporary Flight Lieutenants, Acting Squadron Leaders) O. K. Kewish (256693), 18th June, 1946, R. E. Woods (296330), 24th June, 1946, (Temporary Flight Lieutenants) D. D. Letham (277337), 7th June, 1946, J. B. Foster (257222), M. I. Symonds (257508), 18th June, 1946, W. J. Betts (287464), 19th June, 1946, D. J. Lampard (282874), 20th June, 1946.

The appointment of Temporary Flight Lieutenant J. P. Fleming (263942) is terminated on demobilization with effect from 26th April, 1946.

*Reserve: Medical Branch.*

The following officers are appointed to commissions with temporary ranks as shown with effect from the dates indicated: (Wing Commanders) Ronald William Douglas Fisher (251203), 13th April, 1946, Maximilian Louis Creightmore (291210), 15th June, 1946, (Flight Lieutenant) Howard Ernest Williams (255176), 31st May, 1946.—(Ex. Min. No. 208—Approved 31st July, 1946.)

## Medical Practice.

### NATIONAL SECURITY (MEDICAL EQUIPMENT) REGULATIONS.

### Penicillin Order.

The following penicillin order is published for the information of medical practitioners.

In pursuance of the powers conferred by Regulation 42 of the National Security (Medical Equipment) Regulations, I, Colin Wallace Ross, the chairman of the Medical Equipment Control Committee, acting on the recommendation of the Medical Equipment Control Committee, hereby make the following Order:

*Citation.*

1. This Order may be cited as the Penicillin Order.

*Revocation.*

2. The Penicillin Order, published in the *Gazette* on the twentieth day of September, 1944, is revoked.

*Definition.*

3. In this Order, "penicillin" includes the salts of penicillin, and compounds of penicillin or its salts.

*Supply of Penicillin.*

4. (1) A person shall not supply penicillin (other than penicillin manufactured for veterinary use) to any other person except upon the production of, and for the purposes of, a written prescription of a medical practitioner or registered dentist.

(2) A person shall not supply penicillin manufactured for veterinary use to any other person except upon the production of, and in a quantity not exceeding the quantity specified in, a certificate in the form in the Schedule to this Order signed by a registered veterinary surgeon or by an officer of the Department of Agriculture of a State or the Department of Commerce and Agriculture or the Department of Health of the Commonwealth.

*Exceptions.*

5. Nothing in the last preceding paragraph shall apply to the supply of penicillin

(a) by a wholesale druggist in the normal course of a wholesale dealing—

(i) to a medical practitioner, a registered dentist, or a registered pharmaceutical chemist, for use or disposal in the ordinary course of his practice or business; or

(ii) upon the written order of the person in charge of a hospital or a government department or institution; or

(b) for the Naval, Military or Air Forces.

### The Schedule.

*Certificate for the Supply of Penicillin for Veterinary Use.*

I, the undersigned, hereby certify that the penicillin to be supplied on production of this certificate will be used under my supervision for the treatment of bovine mastitis.

Name of person requiring supply .....

Quantity of penicillin to be supplied .....

.....

(Signature.)

Name and address .....

Qualification .....

Dated this ..... day of ....., 1946.

C. Wallace Ross,
Chairman of the Medical Equipment Control Committee.

## Post-Graduate Work.

### THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

### Lectures in Melbourne.

The Melbourne Permanent Post-Graduate Committee announces that a series of lectures suitable for the M.D., Part II, and M.R.A.C.P. examinations has been arranged. The lectures will be delivered at 4.30 o'clock p.m. as follows:

*Tuesday, October* 8.—"The Management of the Diabetic Patient", Dr. E. Downie, Alfred Hospital.

*Thursday, October* 10.—"The Management of the Diabetic Patient", Dr. E. Downie, Alfred Hospital.

*Tuesday, October* 15.—"Life History of a Case of Mitral Stenosis", Dr. J. Grieve, Children's Hospital.

*Thursday, October* 17.—"The Pathogenesis of Hypertension", Dr. C. Fitts, Royal Melbourne Hospital.

*Tuesday, October* 22.—"The Diagnosis of Coronary Sclerosis", Dr. H. Turnbull, Royal Melbourne Hospital.

*Thursday, October* 24.—"Chronic Non-Tuberculous Infections of the Bronchi and Lungs", Dr. C. Fitts, Royal Melbourne Hospital.

*Tuesday, October* 29.—"Chronic Non-Tuberculous Infections of the Bronchi and Lungs", Dr. C. Fitts, Royal Melbourne Hospital.

*Thursday, October* 31.—"Indications for Surgery in Diseases of the Lungs", Dr. C. J. O. Brown, Alfred Hospital.

*Thursday, November* 7.—"Gastric and Duodenal Ulcer Diagnosis", Dr. D. J. Thomas, Royal Melbourne Hospital.

*Tuesday, November* 12.—"Gastric and Duodenal Ulcer Treatment", Dr. Hurley, Royal Melbourne Hospital.

*Thursday, November* 14.—"Ulcerated Colitis including Differential Diagnosis", Dr. Hurley, Royal Melbourne Hospital.

*Tuesday, November* 19.—"The Use of Thiouracil in the Treatment of Toxic Goitre", Dr. Maxwell, Royal Melbourne Hospital.

*Thursday, November* 21.—"Chemotherapy", Dr. Stanton, Pharmaceutical College.

*Tuesday, November* 26.—"Mechanism of Heart Failure", Dr. J. Hayden, Saint Vincent's Hospital.

*Thursday, November* 28.—"Mechanism of Heart Failure", Dr. J. Hayden, Saint Vincent's Hospital.

*Tuesday, December* 3.—"The Peripheral Circulatory Disorders", Dr. E. Downie, Alfred Hospital.

*Thursday, December* 5.—"Jaundice", Dr. Ian Wood, Royal Melbourne Hospital.

The fee for this course is eight guineas. Applications from those proposing to attend should be forwarded to the Post-Graduate Committee's office, College of Surgeons, Spring Street, C.1, as soon as possible. Those entitled to financial

assistance under the part-time Commonwealth Rehabilitation Training Scheme are requested to indicate this fact on their application.

### Week-End Course at Bendigo.

The Melbourne Permanent Post-Graduate Committee will conduct a week-end course at Bendigo as follows:

*Saturday, October* 12, 1946.—2.15 p.m.: "The Surgery of Jaundice", Dr. Grayton Brown. 8 p.m.: "Common Foot Disabilities", Dr. B. Keon Cohen.

*Sunday, October* 13, 1946.—10 a.m.: "Common Blood Disorders in Children", Dr. J. H. Colebatch. 2 p.m.: "Eclampsia", Dr. A. Hill.

The fee for the course is £1 1s. per day. Medical officers released from the services and accepted under the Commonwealth Rehabilitation Training Scheme part time are exempt from any fees. Those intending to be present should notify Dr. Slater, Honorary Secretary, 18, View Street, Bendigo, at their earliest convenience.

### Lectures and Demonstrations at Melbourne.

The Melbourne Permanent Post-Graduate Committee announces that arrangements have been made to hold the following courses which are suitable for candidates for the D.G.O., Part I, and the M.R.C.O.G. examinations.

#### Bacteriology.

A course in bacteriology by Professor Rubbo will commence on Monday, October 7, at 4.30 o'clock p.m. at the Women's Hospital, and will continue each Monday until December 16. Fee: £8 8s.

#### Pathology Demonstrations.

Demonstrations in pathology by Dr. Bettinger will commence on Wednesday, October 9, at 4.30 o'clock p.m. at the Women's Hospital, and will continue each Wednesday until December 18. Fee: £8 8s.

#### Obstetrics and Gynæcology.

The following demonstrations in obstetrics and gynæcology have been arranged.

*Thursday, October* 10, *at* 4.30 *p.m.*—"Sterility", Dr. J. W. Johnstone, Women's Hospital.

*Thursday, October* 17, *at* 4.30 *p.m.*—"Diseases of the Vulva and Vagina", Dr. R. Fowler, Alfred Hospital.

*Thursday, October* 24, *at* 4.30 *p.m.*—"Evaluation of the Methods of Treatment of Prolapse", Dr. R. Fowler, Alfred Hospital.

*Thursday, October* 31, *at* 4.30 *p.m.*—"Carcinoma of the Uterus", Dr. J. Buchanan, Alfred Hospital.

*Thursday, November* 7, *at* 4.30 *p.m.*—"Genital Fistulæ in the Female", Dr. L. Gleadell, Women's Hospital.

*Thursday, November* 14, *at* 4.30 *p.m.*—"Gynæcological Endocrinology", Dr. J. W. Johnstone, Women's Hospital.

*Thursday, November* 21, *at* 4.30 *p.m.*—"Pelvic Inflammation in the Female", Dr. A. Hill, Women's Hospital.

*Monday, November* 25, *at* 2 *p.m.*—"Acute Surgical Conditions of the Female Pelvis", Dr. L. Gleadell, Prince Henry's Hospital.

*Thursday, November* 28, *at* 4.30 *p.m.*—"Surgical Accidents in the Female Pelvis", Dr. Eccles Mackay, Women's Hospital.

*Thursday, December* 5, *at* 4.30 *p.m.*—"Radiotherapy in Gynæcology", Dr. R. Kaye Scott, Women's Hospital.

*Thursday, December* 12, *at* 4.30 *p.m.*—"Puerperal Sepsis", Dr. A. Hill, Women's Hospital.

*Thursday, December* 19, *at* 4.30 *p.m.*—"Ante-Partum Hæmorrhage", Dr. J. Green, Women's Hospital.

*Tuesday, January* 7, 1947, *at* 4.30 *p.m.*—"Toxæmias in Pregnancy", Dr. A. Hill, Women's Hospital.

*Thursday, January* 9, *at* 4.30 *p.m.*—"Place of Cæsarean Section in Obstetrics", Dr. I. Hayes, Women's Hospital.

*Tuesday, January* 14, *at* 4.30 *p.m.*—"Contracted Pelvis and its Influence on Labour", Dr. I. Hayes, Women's Hospital.

*Thursday, January* 16, *at* 4.30 *p.m.*—"Diseases of the Heart and Lungs Complicated by Pregnancy", Dr. C. H. Fitts, Women's Hospital.

*Tuesday, January* 21, *at* 4.30 *p.m.*—"Anæmias, Thyreoid Conditions and Diabetes in Pregnancy", Dr. Kate MacKay, Women's Hospital.

*Thursday, January* 23, *at* 4.30 *p.m.*—"Manipulative Obstetrics", Dr. A. Wilson, Women's Hospital.

*Tuesday, January* 28, *at* 4.30 *p.m.*—"Diet in Pregnancy and its Influence", Dr. Wheildon, Women's Hospital.

*Thursday, January* 30, *at* 4.30 *p.m.*—"The Use of Blood Plasma and Saline in Obstetrics", Dr. Drevermann, Women's Hospital.

Fee: £10 10s.

Applications from those proposing to attend should be forwarded to the Post-Graduate Committee's office, College of Surgeons, Spring Street, C.1, as soon as possible. Those entitled to financial assistance under the part-time Commonwealth Rehabilitation Training Scheme are requested to indicate this fact on their application.

## THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

### Annual General Course.

The Post-Graduate Committee in Medicine in the University of Sydney announces the following programme.

*Film Programme.*—On Wednesday, October 23, at 4.30 o'clock p.m., in the Stawell Hall, 145, Macquarie Street, Sydney, the following films will be shown: "The Story of Inguinal Hernia" and "The Making of Medical Films".

*Lectures.*—Arrangements have been made for Professor W. W. Greulich, Professor of Anatomy, Stanford University School of Medicine, San Francisco, to give the following lectures: Tuesday, October 29, at 4.45 o'clock p.m., in the Stawell Hall, "Some Observations on the Growth and Development of Normal Children"; Thursday, October 31, at 4.45 o'clock p.m., in the Stawell Hall, "Some X-Ray Studies of the Human Female Pelvis".

All medical practitioners are invited to attend the above programmes, which form part of the annual general course conducted by the Post-Graduate Committee in Medicine, details of which are available from the office at 131, Macquarie Street, Sydney. Telephones: BW 7483-B 4606.

# Australian Medical Board Proceedings.

## QUEENSLAND.

The undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1940*, of Queensland, as duly qualified medical practitioners:

Sisely, Desmond Gerald, M.B., B.S., 1942 (Univ. Melbourne), Thursday Island.

Sampson, Victor Edward, M.B., B.S., 1938 (Univ. Melbourne), Oxley Road, Corinda, S.W.4, Brisbane.

Goyen, Osburn Bracewell, M.B., B.S., 1924 (Univ. Melbourne), Beaudesert.

Atkins, Leonard Arthur, M.B., B.S., 1940 (Univ. Sydney), District Hospital, Mount Isa.

Boydell, William Herbert, M.B., B.S., 1945 (Univ. Sydney), General Hospital, Normanton.

Doneley, Joseph McGlynn, M.B., B.S., 1946 (Univ. Sydney), "Glyndon", Grant Street, Ashgrove, Brisbane.

Hobart, William Robert, M.B., B.S., 1946 (Univ. Queensland), 88, West Street, Toowoomba.

Horsman, Sherwood Arthur, M.B., B.S., 1940 (Univ. Sydney), York House, Melbourne Street, South Brisbane.

Laing, John, M.B., B.S., 1942 (Univ. Sydney), Augathella.

Lehmann, Douglas Keith, M.B., B.S., 1945 (Univ. Sydney), No. 2 Flat, 38, Arthur Terrace, Red Hill, Brisbane.

McGuire, Labeeb Isaac, M.B., B.S., 1946 (Univ. Sydney), General Hospital, Brisbane.

Whyte, George, M.B., B.S., 1943 (Univ. Sydney), 68, Belgrave Street, Manly, New South Wales.

# Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Murray-Prior, Hugh Burnett, M.B., B.S., 1942 (Univ. Sydney), 120, Australian Special Hospital, Little Bay.

Conacher, Dorothy Wolseley, M.B., B.S., 1945 (Univ. Sydney), Rutledge Street, Eastwood.

Neild, William Henry, M.B., B.S., 1936 (Univ. Sydney), A.M.P. Chambers, 55, Hunter Street, Newcastle.

# Obituary.

## JOHN RICHARD HARRIS.

We regret to announce the death of the Honourable Sir John Richard Harris, which occurred on September 16, 1946, at Rutherglen, Victoria.

## JOHN CHARLES WHITE HALLIDAY.

We regret to announce the death of Dr. John Charles White Halliday, which occurred on September 22, 1946, at Wahroonga, New South Wales.

## FRANK McCALLUM.

We regret to announce the death of Dr. Frank McCallum, which occurred on September 25, 1946, at Sydney.

## HENRY PRITCHARD ELLIOT.

Dr. Henry Pritchard Elliot, whose death was announced recently in these pages, was a graduate of the University of Edinburgh, having obtained the degrees of Bachelor of Medicine and Master of Surgery in 1894. He was admitted *ad eundum gradem* in the University of Queensland. He practised in Australia for thirty-eight years. At one time he was government medical officer at Cooktown, Queensland. For the last twelve years of his life he practised at Tuncurry, New South Wales. He was for many years a member of the British Medical Association.

# Medical Appointments.

Dr. J. L. S. Scott and Dr. B. G. Johnston have been appointed honorary clinical assistants to the Surgical Section, Royal Adelaide Hospital, Adelaide.

Dr. H. B. Holmes has been appointed honorary medical officer of the Mount Gambier Hospital, South Australia.

Dr. D. G. Sisely has been appointed Government Medical Officer at Thursday Island and a Health Officer for the purposes of *The Health Acts*, 1937 to 1945, of Queensland.

Dr. K. S. Hetzel has been appointed an honorary physician at the Royal Adelaide Hospital, Adelaide.

Dr. M. E. Chinner, Dr. C. B. Sangster and Dr. J. G. Sleeman have been appointed honorary assistant physicians at the Royal Adelaide Hospital, Adelaide.

Dr. F. D. Rowe has been appointed honorary medical officer to the Port Pirie Hospital, South Australia.

# Books Received.

"Pneumoperitoneum Treatment", by Andrew Ladislaus Banyai, M.D., F.A.C.P., F.C.C.P.; 1946. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Pty. Ltd. 9½" × 6½", pp. 376, with many illustrations. Price: 49s.

"Patients and Appendicitis", by Sir Crisp English, K.C.M.G., F.R.C.S.; 1946. London: J. and A. Churchill Limited. 8¼" × 5¼", pp. 162, with illustrations. Price: 10s. 6d.

"Antenatal and Postnatal Care", by Francis J. Browne, M.D. (Aberdeen), D.Sc., F.R.C.S. (Edinburgh), F.R.C.O.G.; Sixth Edition; 1946. London: J. and A. Churchill Limited. 8" × 5½", pp. 652, with many illustrations. Price: 25s.

"Coast to Coast: Australian Stories, 1945", selected by Douglas Stewart; 1946. Sydney, London: Angus and Robertson Limited. 7¼" × 4¾", pp. 279. Price: 7s. 6d.

"Australian Poetry, 1945", selected by Kenneth Slessor; 1946. Sydney, London: Angus and Robertson Limited. 7¼" × 4¾", pp. 90. Price: 4s. 6d.

"Skyscrapers in the Mist", by George H. Johnston, illustrated by Jack Hanna; 1946. Sydney, London: Angus and Robertson Limited. 8¼" × 5¼", pp. 232, with many illustrations. Price: 10s. 6d.

"Glory and Bondage", by Edgar Snow; 1946. Sydney, London: Angus and Robertson Limited. 8¼" × 5¼", pp. 267. Price: 12s. 6d.

# Diary for the Month.

Oct. 8.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
Oct. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Oct. 8.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Oct. 11.—Queensland Branch, B.M.A.: Council Meeting.
Oct. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Oct. 16.—Western Australian Branch, B.M.A.: General Meeting.
Oct. 17.—Victorian Branch, B.M.A.: Executive Meeting.
Oct. 17.—South Australian Branch, B.M.A.: Council Meeting.
Oct. 22.—New South Wales Branch, B.M.A.: Ethics Committee.
Oct. 23.—Victorian Branch, B.M.A. Council Meeting.
Oct. 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
Oct. 25.—Queensland Branch, B.M.A.: Council Meeting.
Oct. 31.—South Australian Branch, B.M.A.: Scientific Meeting.
Oct. 31.—South Australian Branch, B.M.A.: Council Meeting.
Oct. 31.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 1.—Queensland Branch, B.M.A.: Branch Meeting.
Nov. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 6.—Western Australian Branch, B.M.A.: Council Meeting.

# Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

# Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad per annum payable in advance.